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THE DIAGNOSIS AND MANAGEMENT OF VASO-MOTOR DISTURBANCES OF THE UPPER AIR PASSAGES.*

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It has been known for some years that an individual sensitized to a given proteid may exhibit a characteristic reaction if the proteid is brought in a soluble form in sufficient concentration into contact with a scratch of the skin. In the present connection it is of importance for us to recognize and interpret correctly the significance of this skin reaction. I shall, therefore, briefly describe it and its various degrees of intensity.

The proteid to be tested is applied to a scratch on the skin of the arm. After five to fifteen minutes the positive reactions are indicated by varying degrees of local disturbance. These disturbances of the skin may be ranged in order of intensity as follows: In some cases the first perceptible alteration consists in a sharply circumscribed white area, not elevated, bor-

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dering the scratch for a distance of one to three millimeters. We may find in other individuals the first manifestation to consist of a slightly reddened raised area. In more pronounced disturbances the area of swelling is more extensive, and is more or less white in color, being surrounded by an area of reddening of varying size. When this degree of disturbance appears, it is usually accompanied by itching. It may, in marked cases, attain a considerable size, the edematous area reaching two to three centimeters or more in diameter and surrounded by a centimeter or more of hyperemia.

In the course of thirty minutes, or at most one hour, the reaction fades, and only the slight trauma occasioned by the scratch is left. In the case of certain proteids there may appear on the following day an alteration at the site of the scratch characterized by appearances of inflammation rather than of angioneurotic disturbance. The skin bordering the scratch becomes reddened, slightly elevated and firm. In the course of another twenty-four hours a trace of suppuration may supervene. These disturbances disappear in two or three days. It was at first thought that these inflammatory manifestations were the result of a chance infection, but they have occurred so regularly after testing with certain proteids as to suggest a definite relationship to the proteid substance itself. They follow most frequently tests with staphylococcus p. aureus and albus and streptococcus pyogenes. Among the foods cocoa is apt to show it.

While the occurrence of the skin reaction may be regarded as indicative of sensitization to the proteid employed, yet it is premature to say that a direct relationship invariably exists. Certain observations, for instance, made in the course of hay fever immunization, which I have elsewhere recorded, show that there is not always a skin reaction proportionate to the intensity of the nasal and ocular symptoms. These are matters requiring further study.

During the past two years over four hundred cases showing vasomotor disturbances of the upper air passages were examined by the skin test with different proteids, for the purpose of determining, if possible, the diagnostic value of this reaction, and ascertaining its relationship to the clinical manifestations. For many of these disturbances the special excit-

ing proteid was indicated by the patient's history, as in cases of hay fever, intolerance to fish, horses, egg, etc., but exclusive of the hay fever patients, the larger number gave a clinical history in which the causative agent was unknown to the patient.

Cases of vasomotor rhinitis of recognizable reflex origin, and asthma from cardiac and renal disease are not included. In many instances, particularly of nasal disturbance, the diagnosis of a reflex cause, such as from the genital organs, is difficult to establish, but wherever possible a physical examination from an internist or the family physician was obtained.

CLASSIFICATION OF PROTEIDS.

Recent studies have established a phylogenetic classification of animals and plants, based upon the serum reactions. As the result of the studies, chiefly of Nuttall for animals and of Metz and Gohlke for plants, we are enabled to construct a phylogenetic tree for both kingdoms. The application of these studies to our subject is considerably simplified by our ability to eliminate many members of the animal and plant kingdoms which have no direct relation to man.

Animals.—The primitive proteid molecule in the animal kingdom represented by protozoa exhibits a differentiation in the direction of the molluscs or shell-fish, and passing upward gives off the branch of the crustacea. Above this are the vertebrates, with the branches respectively of fishes, birds, the main trunk terminating in the mammals. In the present connection the mammalian orders which concern us are represented by the domestic animals, and by those which serve as foods, or whose skins as articles of clothing.

Plants.—The phylogenetic tree of the plant kingdom proceeds from the bacteria upward, and divides into the monocotyledons and dicotyledons, the first being represented chiefly by the large family of grasses and by the orders of iris and lily. The trunk of the dicotyledons terminates in the order Compositæ, or family to which the ragweed belongs, and gives off along its course a large number of orders, which have been shown to be of anaphylactic significance, the most important members of these being the nuts, the legumes and the rose family.

PREPARATION OF MATERIAL.

It is of importance to have the test material in the form in which it exists when exciting symptoms. Consequently, we should have it either in the raw or cooked state, respectively, or in both, as the case may be.

It should also be in a stable form, and as pure and concentrated as possible.

This statement is especially emphasized by the fact that we find that different persons vary widely in their degree of sensitization to a given proteid; consequently, one person may react to a proteid dilution of a given strength, while another individual, sensitized to the same proteid, may require a hundred or more times greater strength of the solution to elicit an equivalent skin reaction.

In the case of animals, preparations should be made both of those parts capable of entering the respiratory mucous membrane, as flying scales or hairs, and also of the serum, and of those parts ordinarily ingested, such as egg and milk. Extracts from the epidermal structures are easily obtained by placing scrapings from the skin, hairs or feathers, respectively, in twelve per cent alcohol. Sterilization is accomplished after a few days by the action of the alcohol, and the suspended particles or clear filtrate may then be used. The material may be tested for its sterility, but in no case have I found it contaminated or even after months show signs of decomposition. These extracts appear to keep without deterioration, and I have now two made from horse epidermis, which have been kept at room temperature for over a year, and are apparently as active as when first prepared.

In the case of animal foods, crude but satisfactory material is readily prepared by expressing the juices in a meat press, and subjecting them to rapid desiccation by a blower.

The same method may be followed in the case of plant material. Oils may be removed by leaching with carbon tetrachlorid. Both these animal and plant extracts may then be preserved when thoroughly dry without deterioration.

The proteids of egg and milk can be obtained commercially in the form of a pure powder, as egg albumen and casein, respectively.

While the refinement and separation of the different proteids present in a given material is desirable and represents a

field of important research, yet the above mentioned relatively crude preparations will suffice to answer our first clinical question as to whether the individual is sensitized to the material in question.

In the present work cases were examined both with preparations made along the above lines by Dr. Turnbull in the throat department of the Massachusetts General Hospital, and also by pure refined proteids prepared by Mr. R. P. Wodehouse in the botanical laboratories of Harvard University.

PREPARATION OF BACTERIA.

Suspension of bacteria killed by coagulation of their albumen may be injected intradermally, and the presence or absence of a reaction noted on the following day, as already practiced for a number of diagnostic tests, but I have also found it possible to procure the bacterial proteids in a soluble form, capable of exciting an immediate skin reaction in the same manner as that obtained from the pollen and other plant and animal extracts. At the beginning of the work cultures were grown by Dr. C. G. Page, instructor in bacteriology at the Harvard Medical School, on agar slants, then they were exposed to ether vapor, the object sought being to kill the microorganisms without coagulation of their albuminous constituents. After remaining in ether vapor four days, the colonies were transferred to a twelve per cent dilution of alcohol with water, in order to extract, if possible, the proteids of the bacteria. This alcoholic dilution was chosen, as it had been found by the writer to preserve best the anaphylactogenic properties of pollen and keratin extracts. The quantity of bacteria was about one-third by volume that of the alcohol dilution added; so that, on centrifuging, the bacterial bodies occupied one-third and the fluid two-thirds of the tubes. After a week the fluids were tested on the skin of patients in the same manner as that employed for the diagnosis of plant sensitizations.

Since then a variety of methods of preparing bacteriologic material have been developed by Dr. A. E. Steele of the Massachusetts General Hospital, Dr. I. C. Walker at the Peter Bent Brigham Hospital, Dr. Cleaveland Floyd, and Mr. H. M. Baker at the Harvard Medical School, to all of whom I am greatly indebted for the opportunity which they have given me to test preparations made with this object in view.

In the case of plant pollen, I prefer to keep it in a dry form, as it will then keep unchanged for an indefinite period. I have flowering specimens of grasses and of ragweed in my herbarium, gathered over thirty years ago, the pollen of which still gives pronounced skin reactions in patients sensitized to these plants.

A method was evolved by Mr. Wodehouse for the extraction of ragweed pollen from the flower heads, which enables one to obtain it in a purer form than has hitherto been the case. Plants of ragweed are gathered in the flowering stage and the heads of the flowers stripped off. The habit of this plant is to open the flower heads in succession, and consequently at a given time some will be found to have discharged their pollen and others to be as yet unopened. After drying in air the heads of flowers were washed in carbon tetrachlorid to remove oils, etc., and it was found that most of the pollen came out in the process. Very little solid matter except the pollen will pass through the muslin used for straining. The pollen grains, having passed through, are allowed to dry and are seen to be somewhat paler than those obtained in the natural way, the difference in color being probably due to the removal of lecithin.

The same method is applicable to the other anemophilous plants which furnish a large amount of pollen.

We are thus enabled to obtain the pollen in a relatively pure form, and consequently can make solutions of known proteid strength. Furthermore, this material has not been subjected to the risk of change by treatment with proteid precipitants, and approximates closely to the original proteids in their natural state as they actually reach the exposed mucous membranes.

Experiments were undertaken in regard to preserving pollen and pollen extracts. As I have stated in a previous paper, a dilution of alcohol in water proved most serviceable. The important object to be attained is a solution which will check decomposition with the minimum loss of strength of the active principle. It was found that pollen extract in six per cent alcohol or less showed, after a few days, development of bacteria and proteid decomposition. A dilution of eight to ten per cent was sufficient to check putrefaction, and if the bottle was com-

pletely filled with the solution, it remained free from alteration. If, however, the bottle was frequently opened or left half full, there was a prompt invasion of the yeast organism, with development of vinous odor. This was later followed by the appearance of the mycoderma vini and general turbidity. Solutions from twelve to fifteen per cent could be left open without alteration, and there was no development in them of the mycoderma. These solutions were found to contain an unstable albumin and a relatively stable proteose. The former is readily coagulated by exposure to air, by treatment with alcohol of above twenty per cent, and by heating to sixty degrees C. The proteose, on the contrary, suffers but little on exposure to air, and will endure boiling without precipitation. This observation is in harmony with the familiar fact in wine making that at the close of the first fermentation a clear fluid is present containing the albuminoids of the grape in solution. These are in part thrown down at the first racking and consequent exposure to oxygen, rendering the wine turbid, and requiring often the admixture of isinglass to effect a clarification.

For this reason pollen solutions should be kept as far as possible from exposure to air, and if in the older ones a cloudiness occurs, it should not be filtered, as thereby the albumin would be removed, leaving only the proteose in solution.

The globulins of the pollen grain were separated by Mr. Wodehouse, but were found to be without anaphylactogenic properties.

METHOD OF ENTRANCE OF FOREIGN PROTEIDS INTO THE BODY.

Obviously, under natural conditions the mucous membranes of the respiratory and the intestinal tract afford ready means of access for these substances when inhaled or ingested, and we may consequently speak of such forms of allergy as (1) inhalation or (2) ingestion anaphylaxis.

The possible existence of a third form should be considered, namely, where the symptoms may arise from (3) autolysis of bacterial proteids when in contact with the mucous membranes. In a recent paper I have called attention to this possibility, and have shown that in certain cases of perennial vasomotor rhinitis and bronchitis it is possible to elicit an im-

mediate skin reaction by the application of soluble bacterial proteids to a scratch. Since this paper was written further observations have tended to confirm these views. Such cases are dependent upon the more or less periodic invasion of the mucous membranes by the bacteria to which the individual is sensitized, while during the intervals of infection the patient remains free from vasomotor disturbance.

At least two other possible bacterial causes may exist. In the first instance it is theoretically possible that septic foci, as in chronic tonsillitis, with accumulation of lacunar débris, may be the source of sensitizing proteids; also it is conceivable that the intestinal canal may permit the development of bacteria which may undergo autolysis, and the consequent liberation of proteid material which is anaphylactic for the individual.

The above system of classification is a tentative one, and while for certain portions we may believe that we have sufficiently confirmatory evidence, we are not as yet in a position to accept it as a whole without further investigation.

In the present paper I have endeavored, with this classification as a basis, to study a series of vasomotor disturbances occurring in private practice and in the throat department of the Massachusetts General Hospital.

I.—INHALATION ANAPHYLAXIS.

Under this heading two main types are recognizable, the first being seasonal, the second perennial. In the first instance we find that the symptoms appear at a definite time of the year, and are represented by the very large class of hay fever patients, the symptoms coinciding with the flowering of the special plant in question. The second or perennial form occurs at any time of year whenever the exciting proteid enters the system through the respiratory tract, as in sneezing or asthma from the presence of horses and cats.

I (A)—SEASONAL INHALATION ANAPHYLAXIS OR HAY FEVER.

In this affection recognition of the causative agent is considerably simplified by keeping in mind the following facts: In the first place, our search should be chiefly directed to the monoecious or dioecious plants. As I have previously pointed

out, it is those plants which distributed a large amount of pollen in the air which excite hay fever symptoms. Entomophilous plants which accomplish cross fertilization through the agency of insects are able to be much more economical in the production of pollen, and it is consequently not likely that such pollen can come in contact with exposed mucous surfaces under natural conditions. Such pollen, furthermore, is more viscid and sticky, the object evidently being to enable it to cling to the bodies of the insects which visit the plant in question. It is necessary, on the other hand, for anemophilous plants which depend for their fertilization upon the agency of air currents, to produce a large amount of pollen, and pollen, furthermore, which is relatively light and easily detached from the anthers.

Recognition of these facts enables us to determine with much more certainty the causes of hay fever in a given locality. We may, therefore, pass over the entomophilous plants and confine our attention to the anemophilous representatives. Such are, in this vicinity, certain trees, especially maples and oaks, the grass family as a whole, and certain species of the wormwood family. Although the pines and other gymnosperms produce a very large amount of pollen, yet I have not so far found any instance of hay fever being caused by them. It is in the next place necessary to consider the season of hay fever symptoms. If they occur previous to the flowering of the grasses we shall obviously have to look for the exciting cause in the early flowering shrubs and trees, and among these birch, maple and willow are chiefly responsible. About the middle or the last of May the oak begins to flower in this region, and tests should, therefore, be made for this family. The flowering period of these trees is relatively brief, and consequently the hay fever occasioned by them is less likely to disturb seriously the patient's mode of life.

In the case of grasses we have a flowering period extending from the middle of May in this locality until the first week in July, and the attack of hay fever which they may excite is not only more prolonged but more severe.

Examination of the Compositæ is very much simplified by recognizing that the proteid or active agent of all its members is essentially the same. While minor differences may exist, yet

one active pollen will, for diagnostic purposes, cover the whole order. An interesting example of this is found in two of my patients who spend the winter in Cuba, and who there in February suffer severely from a plant known locally as Escoba amarga, used by the natives to keep fleas from their dwellings, which has been identified for me by Professor Oakes Ames as Parthenium hysterophorus Linn. After the return of these patients to Boston in the early spring, they are free from hay fever until the flowering of the grasses, when a moderately severe amount of vasomotor irritation appears and lasts until the first of July. They then remain comfortable until the middle of August, when severe hay fever appears and lasts until frost. Examination shows that these patients are sensitized to grasses and to ragweed, and that the Cuban plant excites marked skin reaction. Tests of this plant on numerous other sufferers from ragweed who have never been in Cuba excite marked skin reaction in all cases.

Tests, furthermore, of different members of the Compositæ, from the earliest flowering ones, such as coltsfoot, to the late blooming asters, show that with sufficiently strong solutions all ragweed cases will develop skin reactions. Under natural conditions, however, throughout the greater part of the United States, the chief exciting cause among the Compositæ is found in the ragweed, the structure of which will repay examination. It will be seen that the plant is characterized by the possession of long spikes containing the male flowers, and by the localization of the female flowers at the base of the spikes. The amount of pollen which this plant distributes is very large, and it is also extremely light. The flowers open in succession, beginning with the central spike, a few discharging daily—the scattering of the pollen being consequently maintained for a long period. Another plant of the Compositæ which resembles the ragweed in its method of flowering is the Baccharis halimifolia, a favorite plant in shrubberies, although its power of causing hay fever has not hitherto been recorded. The plants are dioecious, and the amount of pollen formed is considerable. The more showy members of the Compositæ, such as field daisy, goldenrod, asters and other garden representatives, possess a relatively small amount of pollen, as well as one of a more generally viscid character. Patients consequently more readily escape, unless the plants are brought into the house.

Examination of the present series of cases has led to the conclusion that the number of proteid types concerned in exciting symptoms is much smaller, at least for New England, than has previously been supposed. During the period preceding the flowering of the grasses, nine cases presented symptoms which terminated by the first or the middle of May, according to the seasons. These patients showed no sensitization to grasses, but reacted chiefly to the maple, birch and willow. Fifty-seven cases showed sensitization to grasses, and one hundred and eighty-three were positive to ragweed and other members of the Compositæ. Sensitization to members of the rose family alone was found in but one case, although a number were found in association with grass sensitization, where it was evident that grasses were the predominating factor.

With regard to the July or midseason forms of hay fever, I believe that with exception of the chestnut and possibly some garden flowers, the chief causes are found either in some special distribution or exposure to either late blooming grasses or early blooming Compositæ.

It is at this season also that the cases of olfactory vasomotor rhinitis or pseudo hay fever are most commonly found. In this affection the fragrance of certain heavily scented flowers excites vasomotor disturbances, ranging all the way from sneezing to actual asthmatic paroxysms. The action of pollen can be definitely excluded by wetting or removing the anthers without influencing the vasomotor symptoms. Skin tests, furthermore, of the pollen show no reaction. The phenomena are plainly reflex, and the path of transmission appears to be along the olfactory rather than the optic nerve. The flowers most commonly responsible are lilies, hyacinths, sweet peas and peonies. The condition occurs either alone, or in association with true hay fever, and is more common in neurotic or impressionable individuals, although not confined to such, several of my cases occurring in highly intelligent individuals, possessing no discoverable neurosis.

It may here be noted that during the hay fever attack a general hypersensitiveness of the patient is apt to appear towards plants for which at other seasons he shows only a minor or no reaction. One must, therefore, be on his guard at this time to observe carefully the relative preponderance or degree of de-

velopment of the various skin reactions, and he will probably be safe in confining his therapeutic endeavors to the plant which causes the most marked disturbance.

I (B)—PERENNIAL INHALATION ANAPHYLAXIS.

(a) Of Plant Origin.—Under this heading I have included those cases of vasomotor disturbance in which the exciting proteid is derived from some other part of the plant than the pollen. Two cases of severe sensitization to flour were found in bakers, who, however, had no hay fever, and who did not react to wheat or grass pollen. These cases, furthermore, could eat bread without disturbance. One case of sensitization to potato was found in a cook. Eight cases of severe sneezing from orris root were encountered in women using face powder containing this as an ingredient. Both the crude root of Iris Florentina and the commercially prepared orris powder produced in these cases marked skin reactions and vasomotor disturbances. In several of these patients the cause of the symptoms had not been previously suspected, but in all instances a complete subsidence followed the discontinuance of the face powder.

(b) Of Animal Origin.—Forty-nine cases were found of sensitization to epidermal scales and hairs of animals or feathers of birds. The symptoms varied from vasomotor rhinitis to severe asthma when inhaling fine suspended particles of the special exciting substance in question. In all cases a definite reaction was elicited by the application of a keratin extract to a scratch of the skin. While the majority were aware of their sensitization, yet for a number of individuals the first recognition of the cause was obtained by the test. This was especially found in the case of children.

Of the various animals the horse was represented by forty-two cases, the dog by eight, and the cat by six. The sheep and cow gave in two cases respectively moderate but apparently definite reaction to keratin extracts from the natural hair, but it did not appear that the first case was affected by the dust from woolen blankets. In two cases definite vasomotor rhinitis was occasioned by the use of feather pillows, and keratin extracts from feathers of the chicken or duck gave well defined skin reactions. The symptoms disappeared on the discontinuance of the feather pillow.

A distinct difference appears to exist between the proteid of the serum and that of the epidermal structures in the animals studied. In the case of the horse, eleven patients gave marked reactions to the keratin extract but no reaction to the serum, either in pure powder or as antitoxin. That this difference is not due to greater strength of the keratin extract employed is shown both by the fact that the majority of patients gave equally marked reactions to both serum and keratin, and by the observation made in one individual of a pronounced cutaneous reaction to horse serum, without a trace of disturbance from the keratin. This patient had had but little to do with horses, but had thirty-eight years previously been bitten by a colt. It is conceivable that in this manner a sensitization to the serum alone may have developed.

While the majority of the dog and cat cases reacted to keratin, both clinically and by the tests, yet two individuals were discovered who reacted to dog serum in a marked degree without sensitization to dog hair. Two similar cases were found reacting to cat serum alone. These four individuals had no discomfort when in the neighborhood of dogs or cats respectively.

Of the two cases of feather sensitization, one showed no reaction to eggs.

These observations indicate the necessity of employing keratin extracts as well as sera when making diagnostic tests of sensitization. Furthermore, the probability is suggested that certain cases of horse fever or horse asthma may in reality receive antitoxin without anaphylactic shock if their sensitization is to the hair and not to the serum of the animal. Finally, the possibility is conversely suggested that the absence of symptoms from horses does not preclude the possibility of an anaphylactic shock from the administration of antitoxin.

II.—INGESTION ANAPHYLAXIS.

Twenty-eight cases were characterized by more or less well defined sensitization to various foods. This number does not represent the true relative frequency of such cases, since their study has been undertaken only during the past six months, and many of the earlier ones may have escaped recognition.

These cases represent all gradations ranging between sensi-

tizations so slight as to have excited no subjective symptoms on the part of the patient to extreme susceptibility, in which contact of the smallest part of the proteid in question caused profound general disturbance. It was noted that the local sensations were referred to the throat rather than to the region of the stomach. Such symptoms were described variously as tickling or itching in the throat, or in more severe cases as swelling and smarting. The general constitutional phenomena were in some cases definite asthmatic symptoms, in others urticaria. Eczema was found associated in a number of instances, and psoriasis occurred once.

Animal foods were prepared for testing, as already described, and instances were found of sensitization to shell fish, crustacea, fish of various sorts, egg, milk and meat. If the skin reaction was marked, the patients were usually aware of their idiosyncrasy. This applies particularly to intolerance to fish and eggs.

Milk was found in one instance to give severe asthma in a child seven years of age. This patient had had more or less constant asthma since being weaned. Physical examination by Dr. J. L. Morse was negative. The skin tests showed reaction to milk, horse and cat keratin. On discontinuing the milk and keeping the cat away, the asthma ceased completely. At the end of two weeks the child was given cow's milk again, with a return of the asthmatic symptoms. This was allowed to continue for a week and the milk discontinued, the asthma simultaneously disappearing. A week later he was allowed to play with the cat, whereupon asthma reappeared. On keeping him away from the cat and giving milk at the beginning in drop doses and gradually increasing, it was possible at the end of six months to establish a tolerance for a glass of milk. The child has during this time been free from asthma.

Cases of sensitization to egg have been referred to children's specialists for immunization by progressive doses, with successful results.

A case of sensitization to raw animal sera was found in a boy twelve years of age. This individual reacted to dog, cat, hog, sheep and ox serum to a high degree. Horse serum and goat serum excited slight reaction. Guinea pig serum gave no reaction. Tests with cooked beef and lamb gave no reaction.

There was well defined sensitization to keratin of dog, cat, horse and lamb, but none to that of the ox. Commercial casein and egg gave no reaction. This case indicates the desirability of having the animal foods prepared for testing under the form in which they are usually taken into the system.

In the case of plant foods we have likewise to prepare the material in the raw, boiled or roasted state, as the case may be. A variety of proteid substances exists in plants, the greater part of which is coagulable by heat. While many proteid substances in aqueous solution require a temperature of 100° C. before being coagulated, others coagulate at 65° C. Proteoses withstand boiling, but may be precipitated by treatment with absolute alcohol. Eleven cases were found of sensitization to grains. In certain of these the patients had no subjective sensations. In four, however, it seemed probable that the asthmatic symptoms of which they complained were attributable to the eating of grains in the form of cereals and bread. Two of these cases were reported somewhat in detail.

Case 1.—G., aged forty years. Asthma since childhood, of unknown origin, more or less persistent throughout the year. No reactions were found to animal foods or to the pollen of grasses, and the patient had no hay fever. Raw and boiled proteids of wheat, barley, oats, corn and rice gave marked skin reactions. Proteids extracted from the inside of bread gave reactions much less pronounced than the raw or the boiled material. Extracts from well browned toast and some roasted grains of wheat, corn and rice gave no reaction whatever. Various breakfast foods were tested, shredded wheat, triscuit, corn flakes, force, Kellogg's wheat biscuit and puffed rice, giving no skin reaction. Grape nuts gave a pronounced reaction. The patient was negative for other plant foods with the exception of beans and peas. Bread and boiled cereals were omitted from his diet for a period of two weeks, during which time his asthma became much less, giving him at the close of this time practically no discomfort except on exertion. On eating again freely of bread, after two days the asthma returned in its original intensity, diminishing later when bread was omitted. When the roasted cereal foods above mentioned, which gave negative reactions, were introduced into the diet, there was no return of the asthmatic symptoms, and the patient is now in greatly improved general health.

Case 2.—S., aged thirty-five years. Asthma many years, more or less constant. Hay fever in spring, moderate. Marked reaction to wheat, raw and boiled, and to bread proteid. Marked reactions to raw and boiled proteids of oat, barley, rice and corn. Positive reactions were obtained also for cocoa and buckwheat. Squash, pea, almond, and Brazil nut gave pronounced reactions. There was no reaction to beet, turnip, cabbage, parsnip, potato, eggs, fish or meat. This case was relieved very much of his asthma on omitting bread and grains, and the other plant foods to which he was sensitized. A proteid extract of wheat was treated with pancreatin for three days. This modified extract caused marked skin reactions. Wheat, rice and corn heated to a temperature of 115° centigrade, gave no reaction, and their proteids on treatment with pancreatin for three days gave no reaction.

These two cases indicate that certain proteids present in the grains are heat stable up to 100° C., but at 115° C. their anaphylactogenic property is destroyed, and is not reintroduced by pancreatic digestion.

Four cases of sensitization to potato, both raw and boiled, were observed. In one of these only was there any systemic disturbance, this consisting in an urticarial eruption. Proteids from baked potatoes gave no skin reaction.

Cases were also encountered of sensitization to nuts, both walnuts, chestnuts and Brazil nuts, some reacting also to almonds, although this is derived from the rose family, and stands far removed in a botanic classification from the others. These cases did not react to the pollen of chestnut or the rose family respectively. A few cases were found of sensitization to strawberries, bananas, oranges and apples in the raw state. Cooking, however, of the strawberries and apples made it possible for the patients to eat the fruits without discomfort. The inference here to be drawn is that the sensitizing proteid was coagulated or destroyed on boiling. The cases of clinical sensitization to grains as a rule had no hay fever, and did not react to the pollen of grasses. It was, however, observed that cases of marked sensitization to grass pollen gave, without exception, a definite skin reaction to proteids, both boiled and raw, of grains. This observation suggests the probability that

the pollen represents the specific plant proteid found throughout all its parts, as Metz and Gohlke have apparently shown, but that in the development of the ovule during the summer other proteids are formed with anaphylactogenic properties differing from those of the specific plant proteid, and that these seed and fruit proteids represent a high degree of differentiation among closely related species. This clinical observation receives confirmation from the works of Wells and Osborne.

These observers have isolated from the seeds of closely allied plants substances resembling proteoses, which are distinguishable from each other. They reach the conclusion that resemblances and differences of these substances are dependent upon the chemical constitution of proteid molecule, rather than upon biologic differences. They, for instance, found that zein, the alcohol soluble protein of corn, did not cause the anaphylactic reaction in animals sensitized with gliadin, the alcohol soluble proteid of wheat, nor with hordein, the alcohol soluble protein of barley. At that time hordein and gliadin had not been tested against one another, but it had been observed that preparations of gliadin from either wheat or rye interacted against one another, as if they were one and the same protein. On testing hordein from barley against gliadin from either wheat or rye, they have since found that these two proteins of different origin also react with one another. Furthermore, they have found that while preparations of gliadin from wheat reacted anaphylactically with glutenin from the same seed, hordein from barley fails to cause reactions in guinea pigs sensitized with glutenin.

It has seemed to me possible that these relationships between the specific plant proteid found in the pollen, and those of the seeds and fruits may be represented graphically as follows: Taking for instance the Rosaceæ, we may let A represent the specific proteid of the family found in the pollen and throughout all the parts of the plant. B, C and D may be taken to denote the fruit proteids of the apple, strawberry and almond, elaborated by the activity of the respective plants during the summer's growth. The proteids present in the fruits will, therefore, be AB, AC and AD. An individual sensitized to the pollen of the rose will react positively to the pollen of all the members of that order, and will also react to extracts from the

fruits or seeds of all. If, however, he is sensitized to proteid B of the apple, he will react neither to the pollen of any of the rose family, nor to proteid C of the strawberry, nor to proteid D of the almond.

This hypothesis has apparently received confirmation from my observations on the Rosaceæ and Gramineæ, as already mentioned.

III.—AUTOLYSIS OF BACTERIAL PROTEIDS.

While the influence of proteids from animals and from flowering plants in the causation of anaphylactic symptoms is sufficiently recognized, yet the part which possibly bacterial proteids may play in these conditions is not yet clear. Clinically we find a large number of cases in which the influence of bacteria seems probable. We may divide these conditions provisionally into acute recurrent and chronic forms. In the first instance we find that the individual goes for considerable periods of time with little or no vasomotor disturbance, and that with the advent of an acute infection of the nasal or bronchial mucous membrane, he exhibits symptoms of vasomotor rhinitis or of asthma. The mucous membranes in these conditions do not show reddening or pus formation which are characteristic of acute rhinitis and acute bronchitis, but present rather the typical picture of hay fever. After the cessation of the acute symptoms, the individual is free from asthma until the occurrence of another infection. The existence of chronic forms of bacterial infection have been recognized by clinicians as being represented on the one hand by chronic sinuitis and on the other by intestinal stasis, and we have probably all had cases where treatment or cure of these conditions has resulted in relieving the vasomotor symptoms. I believe that chronic lacunar tonsillitis with accumulation of detritus must be regarded also as a possible cause of proteid absorption.

In a recent paper I have called attention to the possible explanation of such types of vasomotor disturbance, referring them to the solution or autolysis of bacterial proteids to which the patient is sensitized. On this hypothesis both these acute and chronic forms of vasomotor rhinitis and asthma are dependent upon the occasional or constant anaphylactogenic action of autolyzed bacteria. Experiments which were then reported for the purpose of determining whether bacterial pro-

teids in solution could elicit skin reaction, showed that out of fifty cases positive reactions were obtained from the staphylococcus pyogenes albus, aureus and citreus; also from a bacillus of the Friedlander type, and the micrococcus tetragenus. Since then further study has confirmed these observations, and reactions have been obtained in a number of cases from the bacillus of influenza, streptococcus pyogenes and viridans and pneumococcus, type I and II. It was also observed that after an attack of asthma the skin reaction could not be elicited. but returned again after a few weeks. Much still remains to be done before conclusions can be drawn as to the relation of these organisms to the vasomotor disturbance in question. If it should be confirmed that definite sensitizations to microorganisms exist on the part of these anaphylactic cases, our therapeutic work should receive a highly important assistance. We should, for instance, in the case of acute recurrent infections be guided in the choice and administration of vaccines. The cases of chronic proteid absorption would be more intelligently approached in that we should be led to examine the patient with especial care for the purpose of removing, if possible, the foci of infection, or at least of draining and cleansing the regions in which prolonged contact of bacterial proteids may occur.

ASSOCIATION OF FACTORS AND THE OCCURRENCE OF CLINICAL SYMPTOMS.

A review of the cases has shown that for the majority a variety of sensitizations is present. We find, for instance, often hay fever disturbances from the pollen of different orders, while here and there hay fever patients occur with sensitization also to bacterial proteids, certain classes of foods, or keratin desquamations.

In such cases our task is first to determine the factor which is responsible for the symptoms, and to place in proper perspective the relative influence of the other sensitizations to which the patient is subject. The line of inquiry is usually as follows: First, are the symptoms strictly seasonal? In this case we shall obviously be able to disregard all factors except the various pollens. If the symptoms are perennial, inquiry is next made as to their relation to the occurrence of colds and acute infections. If the answer is confirmative and the patient

states he is entirely free from discomfort except when suffering from a cold or acute bronchitis, we may feel justified in disregarding the influence of both inhaled and ingested proteids, and devote our attention to an examination of his reactions to the various bacteria concerned in acute infections of the upper air passages.

If the symptoms are more or less constant throughout the year, the possible influence of domestic animals should be sought for, especially dogs and cats in the case of children. Nor must the possibility of orris root in toilet or sachet powder be forgotten. Inquiry is next made as to disturbance from different articles of foods, and here the tests should be first made for such substances as enter habitually into the diet: thus, in order of importance, we place eggs, milk, meat, grains, legumes and potatoes, since if the patient is not sensitized to any of these, it is unlikely that the more occasional articles of diet, such as shell fish, fish, root vegetables, greens and fruits are responsible.

If the above tests have resulted negatively, our attention should be directed to the possible existence of chronic absorption of bacterial proteids, and we may then proceed with the examination of the patient as to his sinuses, tonsils and gastro-intestinal tract.

THE MANAGEMENT OF PATIENTS SHOWING HYPERSUSCEPTIBILITY.

In symptoms arising from inhalation of sensitizing substances, if the exposure is likely to continue throughout the year, it is probably wiser to avoid the irritation than to seek to immunize the patient against it. We have at least as yet no record of successful immunization. A case which I reported last year of attempted immunization against horse asthma by the injection of horse serum and keratin extract showed a gain in tolerance to the presence of horses, but on the discontinuation of the treatment the immunity was gradually lost, and the patient has now regained her original sensitiveness. Since for the successful preservation of the required resistance continuation of proteid administration is necessary, and inasmuch as we have yet developed no successful method which the patients would be willing to carry out for an indefinite period, it would seem preferable for the patient to accept his disability, and make the best of it.

Immunization against hay fever by the injection of plant proteids has been described by other writers, and by myself in a previous paper, so that I shall not consider it here in detail. Before, however, we undertake the treatment, we should first decide whether the patient's discomfort is sufficient to justify us in starting him upon a course of procedure which may prove time-consuming and not wholly devoid of risk. The treatment is by no means to be compared to typhoid inoculation, but will require numerous visits, with careful tests of the degree of progress in order to determine the correct dose for each occasion. I cannot too strongly emphasize the importance of this matter of dosage. We are dealing with agents capable in moderate overdose of causing unpleasant shock, and in sufficient amount of leading promptly to a fatal result. The best advice that I can give one starting in this work is to test continually the patient's resistance, and to increase the doses gradually in conformity with the skin or conjunctival tests.

In reviewing the results thus far obtained in three years' treatment of hay fever by hypodermic injection, one must in the first place be guarded in accepting the statements of the patients themselves. As I have previously pointed out, seasons vary, and the physical condition of the patient may likewise differ from year to year. Patients are all too ready to attribute a greater freedom from hay fever symptoms to the treatment employed. It is only where the patient's statements are controlled by clinical tests that they may be accepted as possessing value. With this preliminary statement I will say that the majority of patients who received treatment in the past two summers expressed themselves as satisfied with the results accomplished. From those who felt themselves improved, however, a considerable number should be eliminated, as careful examination of their surroundings, the season and their physical condition might permit other explanations of the diminution in symptoms. The typical individual who presented himself for treatment two or three months before the expected season showed a progressive diminution in the intensity of the skin reactions, and a greater tolerance to the solution employed. In most instances, however, the patients did not escape entirely the hay fever symptoms when the

season came around. The typical behavior was as follows: Hay fever appeared at the usual time or somewhat later, the latter possibly attributable to the season, and reached within a few days nearly its accustomed degree of intensity. This was, however, followed by a striking diminution in the symptoms, so that after the acme of discomfort had been reached, a very considerable degree of relief supervened, and most of these cases remained relatively or entirely free from trouble through the balance of the season. The impression was given in such cases that the amount of treatment which it had been possible to administer in the few weeks before the season was insufficient to effect a complete immunity, but that with the arrival of the season enough pollen was absorbed through the mucous membrane to push the patient, so to speak, over the line. The same results were apparently obtained when treatment was instituted at the beginning of the hay fever season, and in the majority of such instances it was possible to bring about a relief of hay fever symptoms several weeks before relief would ordinarily have been due.

I have received the impression, consequently, that we are not likely in the majority of cases, with the present methods, to do away completely with hay fever symptoms in the course of a single season, but that, nevertheless, sufficient relief may be obtained to make it worth while to continue this method.

Where, however, treatment has been carried on during one season with more or less success, it has been evident that a second course of treatment the following season is more distinctly efficacious.

The usual course of events here is as follows: At the close of the first hay fever season one finds that the patient has attained a relatively high degree of immunity which in the course of the winter progressively diminishes. Beginning again next season, several weeks before the expected date for hay fever, one injects a small dose, carefully determined in the usual way, and follows this by larger doses. In practically all such cases a rapid striking diminution in the intensity of the skin reaction was observed, much more speedy than was the case during the first season. Such cases when the hay fever season arrived were distinctly more benefited than during the previous year, more than half showing no or exceedingly

trifling symptoms. The work is yet too young to enable us to say with certainty what the future will bring forth, but with our present information I should advise considerable caution in the statements to be made to the patients, and especially to explain to them that they may observe little or no benefit during the first season, but that the results obtained lead us to advise a following out of treatment for two years, with the reasonable expectation that the second season's treatment will be more helpful than the first.

Although feeding experiments in the past with extracts of the ragweed do not appear to have been sufficiently successful as to justify their general adoption, yet the work which internists have done with the treatment of egg sensitizations, has suggested the possibility that we may accomplish similar results by administering foods in a natural state derived from the order which produced the symptoms. Experiments were consequently undertaken during the past summer to determine whether the patients sensitized to ragweed pollen would present symptoms from the ingestion of proteid foods derived from the same family.

It was thought that if such internal administration was followed by the development of vasomotor symptoms, either of the skin or of the mucous membranes, an immunization might possibly be accomplished along the recognized lines of treating egg and other food sensitizations.

For this purpose sunflower and lettuce were taken, both being plants of the Compositæ, to which the ragweed also belongs. The pollen of both was found to excite marked skin reaction in cases of autumnal hay fever.

Extracts were made from the seeds of the Russian sunflower which gave positive results, but the intensity of the skin reaction was inferior to that excited by the pollen. The seeds were given to ten hay fever patients, with the direction that they should chew them thoroughly and swallow shortly before their regular meals, with the idea that a certain amount of the proteid might pass into the system directly without digestive alteration. In nine adult cases, however, no gastric discomfort was noted or any influence upon the hay fever symptoms. Oil-free meal from sunflower seeds was prepared which excited a moderate or marked skin reaction, depending upon the

sensitization of the individual. This was administered internally in doses of one to two grams, without exciting symptoms of any kind whatever. One patient, a boy, said that his hay fever was made worse after eating the sunflower seeds.

In the case of lettuce, a strong extract was made from the seeds in a five per cent sodium chlorid solution, which gave a weak reaction. A strong solution of lettuce leaves in a ten per cent sodium chlorid solution (proteins shown to be present by preliminary test) gave a negative reaction.

The amount of proteid present in the lettuce leaf is evidently too small to excite any disturbance, and with one doubtful exception no patients had noted any influence upon their hay fever from eating it in abundance.

It would appear, consequently, probable that we have no foods of the Compositæ order, the ingestion of which is likely to influence the course of ragweed hay fever.

INGESTION.

In the course of this work it has become evident that many individuals exist who show definite anaphylactic skin reactions to various classes of foods, and the problem which here confronts us is to determine to what extent the clinical vasomotor symptoms are dependent upon such a hypersusceptibility. It has in some instances seemed indicated that certain individuals possess food sensitizations, either partially developed or measurably overcome through the acquisition of progressively increasing tolerance, in whom the actual vasomotor explosion is in reality dependent upon some other proteid cause. The following case illustrates this possibility:

D., male, aged forty years. Asthma severe, nearly constant for many years. Marked polypoid degeneration of middle turbinates with involvement of antra. Skin tests were positive for staphylococcus pyogenes albus and a diphtheroid bacillus; negative for pneumococcus, the influenza bacillus, micrococcus catarrhalis and staphylococcus pyogenes aureus. Positive reactions were obtained from proteid extracts of wheat, barley, corn, rice, pea, orange and cocoa. Negative or slight reactions were obtained from egg, milk, beef, mutton, fish, potato, carrot, beet, parsnip, squash and cabbage. Keratin extracts of domestic animals and birds were negative. The patient was

put upon a diet containing only those foods to which he was negative, but without perceptible influence upon the asthma. No operation has as yet been done upon his sinuses, but in view of the above observations this seems the measure most reasonable to undertake.

Conditions of highly developed sensitization to foods have been studied by numerous observers, especially Schloss, Talbot and Chappell. It has been evident that children are much more prone to exhibit anaphylactic phenomena from foods than is the case in adults, a fact possibly due to the greater absorptive power of the gastric mucous membrane in children, or to the acquisition of partial immunity in adults.

If the proteid enters into the accepted articles of diet, experiments have shown that immunity can be obtained in the majority of cases by administering it in small and progressively increasing amount. The details of treatment may be found sufficiently described by the authors mentioned.

The management of cases in which the vasomotor symptoms either demonstrably or by exclusion are attributable to the absorption through the mucous membrane of bacterial proteids is to be undertaken along lines already indicated. Acute recurrent infections may perhaps be checked by the administration of vaccines. The sensitized individual presents here an advantage, in that it may be found possible to select for him with accuracy the special organism required.

Foci of chronic inflammtaion or sepsis should be drained or removed. In three of my cases of long standing asthma, the excision of septic tonsils was followed by entire relief.

Cases of intestinal stasis should be treated by the internist or the orthopedic surgeon, as circumstances may require. Two cases of asthma with ptosis of the viscera were relieved by the application of a proper support.

CONCLUSIONS.

In a large proportion of vasomotor diseases of the upper air passages the disturbances are dependent upon the entrance of a foreign proteid into the system. The method of entrance may be through the contact of the proteid in question with mucous membranes of the respiratory or of the gastrointestinal tract by inhalation or ingestion, respectively. Foreign proteids

may perhaps also develop in or upon these mucous membranes through autolysis of pathogenic or saprophytic bacteria. The application of the skin test to these conditions is of diagnostic value when employed with a recognition of the phylogenetic relationships of animals and plants as determined by studies in serobiology.

Proteid material for testing should be prepared both from the keratin and sera of domestic animals, from the pollen of the chief causes of hay fever, and from the various articles of food, which enter commonly into the diet. Bacterial proteids derived from the various invaders of the respiratory tract should be available either in solution or in the soluble form.

When the skin reactions to the various classes of pollen proteids have been determined, the management of cases will depend largely upon the relative preponderance of the local reactions in relation to the clinical history. If the cause is found to be seasonal, as in hay fever, immunizing treatment by injection of pollen extracts is likely to prove of service. The sensitization returns during the following winter, and treatment must probably be repeated annually. If the cause is perennial and is due to the inhalation of foreign proteids, it is wiser to avoid the cause rather than to seek to effect a cure by immunization. If the disturbing proteid enters into the ordinary articles of diet, a tolerance may be gradually established by feeding the substance in progressively increasing doses. Disturbances of bacterial origin have not yet been sufficiently studied to enable the formulation of a definite plan of treatment, but the results of these investigations confirm our present methods of treatment, and emphasize the importance of draining regions which can retain the products of bacterial activity. Septic foci should be removed. Vaccine therapy is likely in such anaphylactic cases to be more accurately guided than in the ordinary individual.

The results already accomplished in the case of this clinical investigation have led me to the conclusion that we possess in the intelligent application of the skin test a very definite aid in the diagnosis and consequent management of our cases of vasomotor disturbances of the upper air passages. We are as yet only at the entrance of this field, and its exploration must be carried out by the cooperation of laboratory workers in chemistry and serobiology.

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XXVIII.

ANATOMIC RELATIONS OF THE CAVERNOUS SINUS TO OTHER STRUCTURES, WITH CONSIDERATION OF VARIOUS PATHOLOGIC PROCESSES BY WHICH IT MAY BECOME INVOLVED.*

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The object of this paper is to present as clear a picture as possible of the anatomic relations of the cavernous sinus to adjacent structures, and to index in one article, as far as possible, the scattered data of pathologic affections available in the literature of the past fifteen years, together with the important addition of unpublished information obtained from question-letters sent to prominent general surgeons throughout the country. The idea of instituting a search of this kind grew out of the fact that in reading a paper on "Cavernous Sinus Thrombosis and the Future," before the Chicago Ophthalmological Society, November, 1914, the discussion seemed to indicate: First, that while most of us were pretty well informed so far as one or two diseases of the sinus was concerned, our information stopped quite abruptlly at that point; second, that deductions from individual experience was extremely limited; and, third, that possibly statistics with reference, at least to mortality in septic thrombosis of the sinus, might not be accurate. Because of the speculation and the rather chaotic condition of literature, the writer determined to investigate the subject more fully, and to present such matter as could be gathered, even though the results obtained might not prove in the end worth the labor involved. With this preliminary statement, therefore, such material as has been gathered is herein presented.

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ANATOMY.

The cavernous sinuses, two in number, one on each side of the sella turcica,* are a part of the general venous system at the base of the skull. Like the other large venous channels, they are situated between two layers of the dura (splitting of the dura), are lined by endothelium continuous with that which lines the veins generally, and serve to drain the blood anteriorly from about the orbits, and anterosuperiorly from the brain itself. These sinuses carry the bulk of this venous flow backwards through the petrosals to the internal jugular veins, the great outlet for the cerebral circulation. Their anatomic importance depends upon their location in the middle cranial fossa, just lateral to the body of the sphenoid bone, extending from the sphenoidal fissure in front to the apex of the petrous portion of the temporal bone behind, containing venous blood, and being traversed by certain other (transient) structures and being connected internally with each other by communicating small intercavernous sinuses which at the same time surround the hypophysis cerebri.

The cavernous sinuses are so named because they present the picture of a reticulated structure† traversed by numerous interlacing filaments which divide the sinus cavity into irregular compartments. The sinuses are larger behind than in front and measure from two to two and one-fourth centimeters in length, and have a diameter of about one centimeter. A cross section shows these sinuses to be of such shapes as quadrilateral, shallow S outline, or distinctly tooth shaped. As expressed by Piersol[1]: "Its external diameter does not repre-

*Sella turcica (Turkish saddle)—the depression within the three clinoid processes of the sphenoid bone lodging the pituitary body.

†As a clear mental picture of the sinus is important for proper clinical application in disease, it is worth repeating with Halstead that anyone "who has dissected the cavernous sinus will realize that it is not a single cavity, but it is a sinus made up of a number of cavities—from one to fourteen separate cavities, connected by a very small opening, so that you may open and scrape out one of these cavities and still leave others that are infected and continue to spread infection." J. C. Beck has also emphasized the fact that in attempted drainage of the sinus we must take this partition idea into consideration, for in instituting any drainage which might be successful the sinus must be drained as thoroughly as any other thrombotic vessel and that it would be extremely doubtful whether a small slit or puncture in one of these partitions would really accomplish much good.

sent the actual capacity of the lumen, since this is greatly reduced in size: (a) by being traversed by numerous trabeculæ from which fringe-like prolongations hang freely in the blood current, a section of the sinus having very much the appearance of a section of the corpus cavernosum of the penis, whence the name bestowed upon it by Winslow; and (b) by the fact that the internal carotid artery and abducens nerve traverse it, while other cranial nerves are imbedded in its outer wall."

Anteriorly each cavernous sinus receives as tributaries the ophthalmic vein (through the sphenoidal fissure), sphenoparietal sinus, lodged in the dura on the under surface of the lesser wing of the sphenoid, and a smaller cerebral vein (vena cerebri media and sometimes vena cerebri inferior). Anteriorly the sinus is in direct communication with the angular vein at the inner angle of the orbit through the superior ophthalmic vein.

Posteriorly the cavernous sinuses terminate by opening into the superior and inferior petrosal sinuses at the junction of the petrous and sphenoid bones (foramen petrosphenoidale). The cavernous sinus communicates with the lateral sinus by means of the superior petrosal sinus; with the internal jugular vein through the inferior petrosal sinus, and through a plexus of veins on the internal carotid artery (Gray[2]); and with the pterygoid plexus through the foramen ovale.

Internally the two sinuses communicate with each other by means of the intercavernous sinuses (anterior and posterior intercavernous sinuses, with at times a third or inferior intercavernous sinus). The internal surface has anywhere from three to seven openings by which it communicates with its fellow on the opposite side and also with the hypophysis cerebri. On the inner wall within each sinus is found the internal carotid artery, accompanied by filaments of the carotid plexus and the abducens nerve. The communication also between the two anterior ends of the inferior petrosal sinuses at the posterior extremity of the cavernous by the plexus basillaris (or so called transverse sinus) is another anastomotic connection which tends to equalize and distribute blood pressure for the larger cavernous.

The external or outer wall of the sinus is occupied by the oculomotor, trochlear, ophthalmic and superior maxillary

divisions of the trigeminal nerve. All such structures, however, are separated from the blood flowing through the sinus by the endothelial lining membrane continuous with the inner coat of the veins previously mentioned.

Summing up it may be said that the transitional structures of the cavernous sinus number seven in all—i. e., internal carotid artery, carotid plexus, abducens, oculomotor, trochlear, ophthalmic, and superior maxillary nerves.

The upper three-fourths of the external surface of the sinus is in relation to the internal part of the temporosphenoidal lobe, while the lower one-fourth is in relation to the inferior maxillary nerve (mandibular branch).

The superior border of the cavernous sinus is in relation in the posterior four-fifths with the dura covering the cavernous portion of the oculomotor nerve, and in the anterior one-fifth with the lacrimal nerve. The inferior border is said to be "formed posteriorly, by endothelial lining membrane being reflected on the intracranial portion of the nervus maxillaris." (Hoeve.[3])

Relation of Structures in the Cavernous Sinus.—As outlined by Hoeve,[3] in the posterior four-fifths of the sinus, from above downward, the oculomotor, trochlear and ophthalmic nerves rest against the internal surface of the outer wall. The carotid artery, surrounded by the carotid plexus and the nervus abducens, rests against the internal wall, and the plexus cavernosus lies internal to the carotid artery.

In the anterior one-fifth of the sinus the oculomotor nerve and the ophthalmic (first division of the fifth) divide into their branches within the sinus at the junction of the posterior four-fifths with the anterior one-fifth, and this breaking up into branches is the cause of the difference in relations in the sinus and the fissura orbitalis superior.*

***Dissection (from Manual of Dissection and Practical Anatomy of the Head and Neck, Hubertus J. H. Hoeve, edition 1910, pages 347-349):**

(1) Incise dura carefully along the superior border of sinus cavernosus, but do not cut the ligamentum petrosphenoidale at the posterior extremity of the sinus, which bridges over the interval between the apex of the petrosa and the base of the processus clinoideus posterior, and which is covered only by dura in that region.

(2) Reflect the flap of dura outward and downward and expose the interior of the sinus, and by breaking down carefully some of

Variations of the Cavernous Sinus.—According to Quain (as quoted by Mosher): "The cavernous sinus is represented in a child by a venous plexus in the dura mater, which by enlargement and fusion of its channels is converted into the characteristic sinus of the adult. In old age the trabeculæ diminish and the cavity becomes simpler." This is also confirmed by von Lauger,[111] based on corrosion and other preparations from infantile subjects. Instances have been noted where one cavernous sinus is quite rudimentary, leaving practically but one functionating vessel.

Relations of Cavernous Sinus and Transient Structures to Sphenoidal Sinus.—The cavernous sinus in a considerable part of its course overlaps slightly the roof of the sphenoidal cavity and is separated from this air cavity by the thinnest of bony partitions. According to Gibson,[4] from an examination of eighty-five sphenoidal sinuses, the roof of this sinus varies from 0.25 millimeter to two millimeters, averaging approximately one millimeter in thickness. Bony dehiscences have been observed by J. C. Beck and others between the sphenoidal sinus and carotid canal, in which the internal carotid artery might easily have been injured by too forcible instrumentation in the sphenoidal cavity. In most specimens the casing of the bony carotid canal is plainly seen bulging into the cavity of the sphenoid sinus near the posterior external angle. For the most part, therefore, it may be said that the cavernous sinus occupies a fairly constant position above and lateral to the body of the sphenoid bone (junction of roof and external wall of the sphenoidal cavity), and given a large sphenoid cavity, not only will the cavernous sinus be almost in contact, but also with any kind of a posterior prolongation in the body of the bone the basillar plexus may be in relation to the sphenoidal sinus as well. Indeed, diverticula are so frequently present in many sphenoids examined that through them quite a part of the circumference of the cavernous sinus region may be affected, bringing at times most all of the transient nerve trunks of the

the trabeculæ find the nervus abducens and the sinus petrosus inferior as they pass through the foramen petrosphenoidale inferior to the ligamentum petrosphenoidale.

(3) Find that all the structures passing through the sinus are covered by the lining epithelium of the sinus, and so not in contact with the venous blood.

cavernous sinus or even the Vidian canal (Sluder[5]) and Gasserian ganglion (Mosher) in relation to the sphenoidal cavity.

CONSIDERATION OF INDIVIDUAL STRUCTURES.

Intercavernous Sinuses (Circular Sinus).—The circular sinus (anterior, posterior and frequently inferior intercavernous sinuses), which connects the two cavernous sinuses, is found in the sella turcica, surrounding the hypophysis cerebri and within the attached border of the diaphragma sellæ. It is formed most often by the three sinuses mentioned: the anterior intercavernous sinus, usually the largest, passing in front of the pituitary body; the posterior intercavernous sinus behind, and the inferior intercavernous sinus below this body. These sinuses receive branches from the pituitary body and from the dura mater. The intercavernous sinuses, together with that portion of the cavernous sinus between their terminations, form what is usually termed the circular sinus, although in reality it represents only a part of the extensive venous system around the body of the sphenoid bone.*

Basillar Sinus (Plexus basillaris—Transverse Sinus).—The basillar plexus consists of several interlacing veins resting on the dorsum sellæ (mostly basillar process of the occipital bone), connecting the two anterior ends of the inferior petrosals, close to the posterior extremity of the cavernous sinus. They lie between the posterior clinoid processes and the anterior margin of the foramen magnum, and communicate also with the anterior spinal veins. The basillar plexus can be detected by its bluish color when the dura is carefully removed in this region. Like the cavernous sinus, the basillar sinuses[111] are persistent channels of what was originally, in the fetus, an extensive venous plexus.

Superior Petrosal Sinus.—The superior petrosal sinus lies along the superior border of the petrous portion of the temporal bone (superior petrosal sulcus), in the anterior part of

*The two middle cranial fossae are connected with each other by the sella turcica, which contains the hypophysis cerebri and is bridged over by the diaphragma sellæ. The little oval opening in its middle is the incisura diaphragmæ sellæ, through which passes the infundibulum. The entire hypophysis is surrounded by venous sinuses (intercavernous sinuses) located in the walls of the hypophyseal fossa.

the attached margin of the tentorium cerebelli. It is small and narrow and serves to connect the posterior extremity of each corresponding cavernous and the bend or knee of the lateral sinus. It is a little above the petrosphenoidal foramen and trigeminal nerve. It receives some cerebellar and inferior cerebral veins, and veins from the tympanic cavity. According to Page,[23] the superior petrosal is frequently found to empty itself into the lateral sinus, not by a single opening, but through a series of openings three or four millimeters apart, extending along the sinus wall at a distance of two or more centimeters.

Inferior Petrosal Sinus.—The inferior petrosal sinus, larger but not as long as the superior, forms the shortest connections between the cavernous sinus and internal jugular vein. It is situated in a groove (inferior petrosal sulcus) formed by the petrooccipital suture. The inferior petrosal passes anteriorly through the foramen petrosphenoidale along with the abducens nerve, and behind through the anterior compartment of the jugular foramen, ending in the commencement of the jugular vein. The anterior ends of the inferior petrosals are connected with each other by the interlacing vessels of the plexus basillaris. The inferior petrosals receive as tributaries some inferior cerebellar veins, veins from the internal ear, pons and medulla.

Sphenoparietal Sinus.—The sphenoparietal sinus and ophthalmic veins may be regarded as the anterior beginning of each cavernous sinus. The sphenoparietal sinus is lodged in the dura below the inferior surface of the lesser wing of the sphenoid. It is located above the oculomotor nerve, and has as tributaries small veins from the diploe and the middle meningeal.

Ophthalmic Veins.—(a) The ophthalmic veins, superior and inferior, according to Gerrish,[6] have no valves, and the blood, under certain conditions, may flow from behind forward into the angular vein or its branches, and thus obviate pressure in the veins of the retina when the cavernous sinus is obstructed. The superior ophthalmic, beginning as the nasofrontal, communicates with the angular at the inner angle of the orbit. It also anastomoses with the inferior ophthalmic. Forming a short single trunk, it passes through the inner extremity of the sphenoidal fissure to the cavernous sinus. The swollen end of

the superior ophthalmic, just before it enters the cavernous, has been dubbed sinus ophthalmicus.

(b) The inferior ophthalmic vein receives veins from the region of the floor of the orbit, and either passes through the sphenoidal fissure to join the pterygoid plexus of veins, or more directly backward through the sphenoidal fissure to the cavernous, either separately or by forming a common trunk with the superior ophthalmic.

Emissary Veins.—The emissary veins are vessels which pass through foramina in the skull wall to connect the cranial sinuses with the veins of the diploe and veins external to the brain (as in and about the scalp). These are important, in that they may serve as possible channels along which infection may be carried occasionally from the surface to the interior of the cranium. They are not always constant as to presence or location. The cavernous sinus, through emissary veins, is connected with the pterygoid plexus through the foramen ovale and Vesalius, as well as through the inferior ophthalmic vein; with the pharyngeal plexus through the foramen lacerum medius, and with the internal jugular vein by the carotid plexus through the carotid canal (Gerrish[6]).

Carotid Artery.—The second or petrous portion of the carotid is within the carotid canal, while the third or cavernous portion is that part of the vessel lying within the cavernous sinus along the side of the sella turcica. The artery first enters the bony carotid canal in the petrous portion of the temporal bone, and follows its windings, first upward a short distance, curving forward and inward, and again ascends to enter the cranial cavity in its cavernous portion. In the cavernous portion the artery follows the carotid groove or sulcus along the lateral body of the sphenoid extending from the posterior clinoid process behind to the anterior clinoid process in front, at which latter point it again curves upward and slightly backward to perforate the dura forming the roof of the sinus. After piercing the dura it passes between the optic and oculomotor nerves and runs to the internal extremity of the fissura lateralis cerebri, where it finally divides into terminal branches. Just at the point where the artery turns backward and internal to the anterior clinoid process, it may be easily mistaken for the ophthalmic artery (Hoeve[3]). The abducens nerve passes

along with the carotid in the cavernous portion and external to it. Sympathetic filaments from the carotid plexus also pass with the carotid artery. In the cavernous portion the distribution of the carotid branches are as follows: Few small arteries to the hypophysis cerebri, Gasserian ganglion, arteriæ receptaculi, anterior meningeal and ophthalmic. According to Piersol,[1] where the internal carotid enters the cavernous sinus at the internal orifice of the carotid canal, the sinus projects downward around the artery in a funnel-shaped manner, and from it there arises a close network of veins, the carotid plexus or carotid sinus, which completely invests the artery throughout its course through the carotid canal, at the lower opening of which it is continued into one or two veins which open into the internal jugular.

Carotid Plexus.—The carotid plexus of nerves, situated on the outer side of the internal carotid artery, communicates with the Gasserian ganglion, sphenopalatine ganglion, sixth nerve, Jacobson's nerve, glossopharyngeal, and distributes filaments to the wall of the carotid artery and to the dura. Filaments of the carotid plexus may occasionally form a small ganglion (carotid ganglion) on the under surface of the artery. The relationship of these portions of the sympathetic nervous system to other adjacent structures of this region has been well worked out by Haskins.[7]

Cavernous Plexus.—The cavernous plexus of nerves is located below and internal to the carotid artery within the cavernous sinus. It communicates with the third, fourth, ophthalmic division of the fifth, sixth, ophthalmic ganglion, besides distributing filaments to the wall of the carotid artery. It is formed chiefly by the internal division of the ascending branch of the superior cervical ganglion. Its physiology belongs to the physiology of the sympathetic nervous system.

PATHOLOGIC AFFECTIONS OF THE CAVERNOUS SINUS.

The following data relating to diseases of the cavernous sinus have been obtained from question-letters sent to members of the American Surgical and Western Surgical Associations, and from the scattered monographs of affections available to me in the literature of the past fifteen years. While the information is not especially voluminous, it may serve as

a basis to build upon in attempting the study of the cavernous sinus as a distinct and, one might say, separate organ of the body. As illustrating the difficulty and disappointment in gathering material, I would quote an extract from a letter from Dr. Louis B. Wilson, director of laboratories at the Mayo Clinic, Rochester, Minnesota, who reports that "No specimen, either from operation or autopsy, showing pathologic affections of the cavernous sinus has come into the laboratory since I have been connected with it, now ten years."

Out of two hundred and eighty-four letters mailed to members of the American Surgical and Western Surgical Associations, one hundred and twenty of the men responded, but of the number responding only forty-four were able to cite cases either directly or indirectly concerning the sinus. The classification of affections from the replies received from these letters is as follows: (1) Septic thrombosis, (2) gun shot wounds, (3) involvement from fracture of the skull, (4) arteriovenous aneurism, (5) injury during miscellaneous operations, (6) involvement by malignant growths, (7) congenital affections. To this from the literature gone over must be added (8) marasmic or socalled aseptic thrombosis and for the present (9) intermittent exophthalmos, both of which last are rare conditions. The foregoing admits, therefore, of a classification of nine separate and distinct parts, all worthy of attention, and is so far as I know practically the first attempt of its kind to systematize, and it is only a beginning.

(1) SEPTIC THROMBOSIS.

This subject has been pretty well thrashed out of late, so far as the ordinary avenues of infection are concerned, and the few case reports yearly do not add a great deal to our present knowledge. The principal question at issue concerns itself with early reasonably positive diagnosis, and whether successful treatment by direct operation with the many difficulties which attend, is feasible or not. So far, attempts in this direction have rather dismally failed.

From the question-letters sent out, which means mostly unpublished information, twenty-nine cases were reported to me of septic thrombosis, with recovery in three cases (Scholz, Kanavel, Halstead—see Table 1), or 9.6 per cent. Of these

cases of recovery, one from the ear recovered through energetic and thorough aural surgical procedures alone—i. e., radical mastoid and jugular ligation operation; another, neck infection, recovered through local neck drainage, and the third, complicating an ethmoid operation with syphilis, got well on expectant, purely medical treatment. A direct operation was not attempted by any one of the twenty-one general surgeons upon whose data part of this report is based. (Table 1.)

In forty-four cases of septic thrombosis selected from the index of literature here appended (Johnson,[29] Adair-Dighton,[38] Bircher[107]) three recovered, or a percentage roughly of seven. Jackson[54] also gives a percentage of recoveries of about seven per cent, while Dwight-Germain[104] place their recoveries in a total of cases looked up as 7⅓ per cent, which seems to be not far out of the way and settles, I think, from the statistical point, that a patient does have some chance to recover, which some of us have scarcely thought possible.

To continue the subject farther. While it is not my purpose to present a too lengthy discussion of this topic, this article would be incomplete without some general consideration of the condition and some statement as to just how thinking men are beginning to view the matter of septic thrombosis about the body of the sphenoid bone. As has been alluded to elsewhere, physicians have felt and are feeling the need of more definite moral support, both in the matter of fairly early reasonable positive diagnosis, as well as from the standpoint of any line of treatment which might be contemplated when confronted with this disease. It may be mentioned also at this time that the problem of septic thrombosis is a borderland one, belonging to various fields, with, however, the striking features chiefly ocular. In consultation, therefore, we should, first of all, include the ophthalmologist for his eye findings, even though the actual charge of the case be placed in the hands of the general surgeon doing a good deal of head or pituitary body work, or of the rhinootologist specializing on the anatomy of the sphenoid region. This certainly, with some acquaintance with literature, would probably provide the best basis to work upon for intelligent future investigation.

Rarity of Cases.—The study of cavernous sinus thrombosis has been limited in the past (1) because the cases are rare and

the average physician, even in a lifetime, will scarcely see more than two or three sufferers with the disease; (2) being surgically less approachable perhaps than almost any other structure in the body, few men really consider the sinus available for surgical interference, especially when the patient may seem already in a septic moribund condition and the cavernous sinus itself only one of a number involved.

Etiology.—For practical working purposes we may consider infection of this sinus as arising from two general directions: 1, posteriorly from about the mastoid or ear region from a thrombosed lateral sinus, the infection spreading directly forward against the blood current along one of the petrosals; 2, anteroinferiorly and a little less common by the infection from a carbuncle or wound of the face, jaws or nose, being carried backward, upwards and internally to the interior of the cranium along the drainage system of the ophthalmic vein or by anastomozing veins directly upward from about the throat. The origin of infection at a remote region of the head or neck is one of the most important and tangible clues in diagnosis.*

Symptoms for Reasonably Early Positive Diagnosis.—The very first question that confronts us is the necessity of making a reasonably positive diagnosis fairly early in the disease, so as to make it worth while. As the findings of the ophthalmologist are always awaited with a good deal of expectancy by the other consultants, any opinion of value must needs show considerable acquaintance with the literature, a watchful lookout for just such a possibility under certain conditions, and, most important of all, getting every bit of information which is possible to obtain in any way about the patient before rendering a decision as to what is clearly taking place. Since the later classical signs with which many of us are familiar are among nature's last efforts, not until we have the most careful analysis of earlier symptoms, combined with almost intuitive judgment, can we expect to improve in diagnosis—and improve we must, if we would expect hopes of any future successful line of treatment. While medical men are naturally averse

*The literature of septic thrombosis is well covered (successive articles) in numbers 104, 72, 68, 54, 37 of the index and the present paper.

to taking the positive side in medicine, the fact that so many physicians stand by without feeling sure of their ground until autopsy, even after unmistakable symptoms are presented, often a number of days before death, makes the positive position for the skilled diagnostician all the more necessary just at present. One thing perhaps that has caused a good deal of confusion has been variations in the early onset of the disease, according to the origin of the infection, and also variations due to the intensity of infection.

So many factors enter into the causation of earlier symptoms that with the exception of beginning exophthalmos and chemosis of the lids of one eye, any one or often several of other signs, such as strabismus (internal or external) from paralysis of one of the nerves in the sinus wall, mild bilateral fundus congestion or marked papillary edema, meningeal symptoms, etc., may at first be absent. In a general analysis of cases the order of appearance of symptoms is about as follows: Deep seated pain about the eyes, septic temperature, varying widely with respect to degree, slight orbital congestion, lacrimation, conjunctival edema, a little lid swelling, slight ptosis, and beginning exophthalmos. The foregoing, associated for the first time with a more distant infective focus about the head or neck, at once suggests that all is not well, and that thrombosis of the cavernous sinus is developing. Difficulty in swallowing, neck tenderness, and some swelling on the infected side may also be present in certain cases and absent in others. Increasing lid edema and exophthalmos very quickly presents a not-to-be-forgotten picture. After thirty-six or forty-eight hours, beginning involvement of the opposite eye by lid chemosis and exophthalmos is noted, which is easily seen to come from behind and not to have jumped across the bridge of the nose, and is so characteristic as to admit of little argument in diagnosis. Pupillary changes, marked papillary edema, meningeal or brain abscess symptoms, metastasis in a distinct joint, septicemia and death is the final culmination.

In making a diagnosis of a given case, even without previous experience, one may formulate this rule: When we have increasing exophthalmos and lid edema on the same side as a somewhat distinct infecting septic focus, any edema of the face and neck, septic temperature, and the slightest evidence

of beginning lid edema or exophthalmos of the opposite eye, we are safe in saying that the later positive symptoms of the cavernous sinus thrombosis are present beyond a doubt. A fairly early diagnosis in cases where the infection originates posteriorly about the ear or back of the neck is comparatively easy, and should present no difficulty. Cases arising anteriorly about the face, nose or jaws, however, present at times considerable difficulty, especially if the focus of infection is in or very close or the orbit. In some instances where the trouble started anteriorly even, yet owing to our lack of individual clinical experience, it is practically impossible to be certain of the diagnosis until the second or opposite eye begins to show some slight involvement by lid chemosis and exophthalmos. It is in these instances, however, where intuitive judgment points out what might very well happen under suspicious circumstances, as much as any reliance upon ophthalmoscopic evidences of bilateral venous stasis within the eyeball. While the condition is to be differentiated from septic infection of the frontal sinus, beginning erysipelas, orbital cellulitis and a few sphenoidal conditions, in reality these should be ruled out without much difficulty.

Prognosis.—The prognosis is bad, and the mortality for all working purposes, under present conditions, should be considered much higher than our statistics of seven per cent would indicate. Indeed, for all practical purposes in an individual case we have little reason to hope for a recovery. Medical science has not as yet attempted a systematic campaign to solve the problem of earlier diagnosis or practical treatment. In literature of recent years and from private sources (Table No. 1, and References Nos. 29, 38, 107) I have obtained six instances where a diagnosis of septic thrombosis of this sinus has been made and nature was able to overcome the infection and the patient lived. In these instances attention was given solely to the original infecting focus, and not to the cavernous sinus itself. Of seven further attempted operations of any kind to drain the sinus by direct surgical procedure (Bircher,[107] Voss,[108] H. Knapp,[105] Dwight-Germain,[104] C. A. Ballance, Ballance-Hobhouse,[37] and Mosher,[25] all have failed to accomplish the purpose, and two at least we know failed even to get to and open the sinus toward which the operation was directed.

In correspondence last year with five each of our prominent general surgeons, pituitary body workers, otorhinologists, anatomists and neurologists, eleven reported no personal experience with the disease and would make no comments; five believed the sinus might be amenable to treatment, and nine were most skeptical and expressed themselves as against any operative procedure or real hopes for the future along this line. As possible operative routes to attack the sinus or methods of treating same the following were mentioned as still to be perfected: Mosher's proposed anterior route through the orbit, Hartley-Krause[112] Gasserian ganglion route through the side of the skull, Luc[81]-Langworthy[68] routes upward through one side of the nose and sphenoidal cavity, and lastly, in certain otitic cases, energetic treatment of the original focus alone from behind—namely, by opening the lateral sinus and jugular bulb thoroughly and resecting the internal jugular vein.

Recognition of Factors Necessary for Real Progress.—From the foregoing it must frankly be stated that no substantial support can be deduced from past experience. What little there is in literature is all against expected saving of life by any special operative endeavor on our part beyond thorough operative exposure of the site of original infection. In reality, reports would seem to indicate that such cases "represent organisms which are ruined and in which all science loses its right to interfere." In the face of this, however, the few men who have studied the question most thoroughly and attempted something in the way of relief seem loath to accept such an unalterable dictum, but cling to the hope, even though not very well founded, that little will be lost by following a more active and logical operative plan until future systematic demonstration of failure—not in one or two scattered instances, but from dozens of attempts made all over the world and under all kinds of conditions—has settled the question more finally. It certainly is time, if we are to progress in our handling of so baffling a disease, that we must tenaciously adhere to three cardinal points connected with it: (1) Standing flat footed upon a reasonably positive diagnosis as early in the disease as possible; (2) viewing the sinus as we would any other large vessel which has become thrombotic; (3) following routinely that operative path which would seem to the surgeon in charge

as best fitted for drainage. This last will naturally vary, operators preferring methods as selected and reported by themselves.

Limitations of Deductions From Purely Cadaver Work.—Last year the writer endeavored by systematic work on the cadaver to once more approach the matter of practical anatomic access in as unbiased a manner, and choose, if possible, the best, if one be here allowed such a term, of the three routes offered so far—i. e., (a) Hartley-Krause Gasserian ganglion route through an osteoplastic resection of the side of the skull; (b) Mosher's proposed anterior orbital exenteration and chiseling through the orbital plate of the great wing of the sphenoid; and (c) the nasosphenoidal route as outlined by Langworthy,[68] but which the pituitary body workers alone have indirectly demonstrated as containing any real practicability on a living subject. From the foregoing it may be stated that the surgeon, having specially prepared himself for the operation, is able to tap the cavernous sinus from any one of the three above directions, but not by any means easily. Under favorable conditions my own selection, in addition to thorough opening of the original focus of infection, would certainly be as follows, if given an actual case:

Writer's Proposed Operation.—Light ether vapor anesthesia. Plugging of posterior nares on one side and free injection of adrenalin chlorid solution about the operative region. Quick removal of obstructing ethmoid labyrinth, middle turbinate and anterior sphenoidal wall by the use of ethmoid curette, turbinate forceps, sphenoidal curette, punch, and long narrow-handle gouge and hammer. Once in the sphenoid cavity the author's straight and angular blunt curettes with overhanging edge[68] can be pushed through the roof of the sphenoidal sinus, close to the junction of its roof and external wall. The blunt ends of these curettes will push the carotid artery aside without damage, and by rotating the spoon in a forward direction away from the carotid artery, the overhanging edge of the curette catches bone and a hole of some size can be made leading directly into the sinus. This can be further enlarged by use of curettes, assisted, as mentioned, by a long narrow chisel and hammer. The chief danger of the operation here, I would say, is not quite so much the thick walled large carotid artery,

but rather some unexpected small branches given off frequently in this region which when accidentally torn by instrumentation might prove troublesome or opening only a partially thrombosed sinus—much of this, however, is at present pure speculation.*

Cavernous Sinus Thrombosis and the Future.—In concluding the subject of septic thrombosis, I would repeat that the future alone must determine the real practicability of attempted earlier diagnosis and direct operation. At the present time, and looking fearlessly into the future, it would seem wise, until more evidence to the contrary is produced, to look upon these cases with increasing hopefulness and as offering at least occasionally surgical opportunities which should not be ignored.

(2) GUN SHOT AND OTHER PENETRATING WOUNDS.

Eight cases of gun shot wounds were reported by letter in which the sinus had been penetrated and otherwise badly injured. Six cases died, and two (Raymond, Halstead) recovered. (Table 2.)

(3) INVOLVEMENT BY SKULL FRACTURE.

Fracture at the base of the skull, with moderate laceration

*Luc, of Paris (1905), proceeded by making a large opening through the anterior wall of the maxillary sinus on the same side as the cavernous sinus which he wished to open, and another smaller one into the nose, to locate the anterior wall of the sphenoid. Using a straight chisel, however, he was unable to penetrate the lateral wall of the sphenoidal cavity on that side, and so came to the conclusion that the cavernous sinus should be approached through the maxillary antrum on the opposite side of the head. This gave him the desired angle (oblique), and he was able on the cadaver to open the side of the sella turcica and penetrate the sinus without injuring the important structures. Brunert also seems to have had a clear idea of some time opening the cavernous sinus through its relationship with the accessory sinuses of the face. My own interest in this subject dates from seeing my first case in 1904, and feeling that the sinus might possibly be drained (gravity method) by way of the sphenoidal cavity through the same side of the nose. J. C. Beck, in 1912, in investigating the subject of approaching the hypophysis from various routes, developed a method of reaching the hypophysis by way of the antrum following the Jansen operation, and operated successfully two cases of hypophyseal tumor. From cadaver work, having in mind also at the same time the cavernous sinus, both Beck and J. R. Fletcher emphasize the tremendous difficulty of reaching this sinus through the nose. At best, certainly any kind of an operation will many times be seen to be a useless proposition under some conditions.

or complete rupture of the sinus, was reported in six cases. Two cases (Ochsner, Bevan) recovered, one by tamponing the sinus and the other by expectant treatment with, however, the final loss of the eye itself. (Table 3.)

(4) ARTERIOVENOUS ANEURISM (RUPTURE OF INTERNAL CAROTID ARTERY INTO CAVERNOUS SINUS).

Fifteen cases of rupture of the internal carotid artery in the cavernous sinus, producing arteriovenous aneurism with pulsating exophthalmos, for which ligation of the common carotid or internal carotid became necessary, were reported by letter. Improvement or full recovery ensued in twelve, death in three cases. The principal causes as given were syphilitic weakening of the wall of the artery and traumatism as by fracture or a blow on the head. In one of these cases (Halstead) thrombosis of the cavernous sinus took place after ligating the internal carotid artery with, however, good recovery from the cavernous sinus thrombosis. (Table 4.) Rupture of the carotid into the cavernous has been extensively reviewed in literature the past few years.

(5) INJURY TO CAVERNOUS SINUS DURING MISCELLANEOUS OPERATIONS.

Three cases were reported by letter in which the cavernous sinus was quite badly injured—two during a Gasserian ganglion operation with, however, good recovery, and one during an hypophysis operation (Halstead), with death from intracranial hemorrhage. In the literature appended, Emerson[62] reports serious injury of the sinus with death from curettement of the sphenoidal sinus. Cushing[82] reports a case of injury to the sinus during a Gasserian ganglion operation, followed by cavernous sinus thrombosis and recovery. The attention which the pituitary body is receiving, with operative attempts at treatment, must surely in time throw much more positive light on the cavernous sinus and its transient structures.

(6) MALIGNANT GROWTHS INVOLVING CAVERNOUS SINUS.

Two cases of sarcoma with death (Benjamin) were reported in my list of question-letters, but reported without any

further data. In literature appended, Knapp[105] reports a case of orbital sarcoma followed by aseptic thrombosis of the cavernous sinus, with successful sinus operation by Hartley, in which the patient lingered for over two months. Macewen[102] mentions a case of septic thrombosis and death from carcinomatous involvement.

(7) CONGENITAL AFFECTIONS.

One congenital growth (fibrocystic) was reported by Veer (Table 7), in which the zygoma and malar bones were resected in order to reach the area, and many vessels surrounding the cavernous litigated. The patient made a good recovery. I have not obtained as yet a detailed report of this case. One might well expect here also the possibility of some of the rarer forms of congenital angiomata deep within the orbit, in which a correct diagnosis is difficult, to involve the cavernous sinus in some of its ramifications.

(8) MARASMIC (ASEPTIC) THROMBOSIS.

No cases of purely marasmic thrombosis were reported in the question-letters sent out. A few instances are on record in literature, however, occurring during the course of debilitating or marasmic diseases, such as pregnancy, typhoid fever, of socalled marasmic thrombosis of this vessel. Zentmayer-Weisenburg[73] report such a case associated with menstrual disturbances and pregnancy, and Knapp[105] presents a case of aseptic thrombosis from sarcoma of the body of the sphenoid bone and apex of the orbit originating in the cavernous sinus.

(9) INTERMITTENT EXOPHTHALMOS.

This is a most interesting condition, new, I think, to most of us, and its exact relationship to the cavernous sinus, if any, still a little unsettled. For the present, as mentioned, it seems permissible to mention it here.

No cases of this affection were mentioned in the question-letters, so that the literature of the subject had to be consulted for the information. True intermittent exophthalmos, a rare condition, is fully reviewed by Posey[80] and Alger,[75] and must not be confused with pulsating exophthalmos, which last is due to rupture of the internal carotid artery in the cavernous sinus.

In a case of true intermittent exophthalmos, owing to changes in the orbital veins, one eye will be pushed forward out of the orbit, but quickly recedes as soon as venous pressure in the orbital veins and cavernous sinus becomes normal. In a typical case the eye proptoses in a few seconds whenever the head is in a dependent position, as in stooping, or whenever a venous congestion of the cranium is produced by a great muscular exertion or by a local pressure on the neck. The explanation of the phenomenon seems to be that under certain conditions which produce increased muscular tension in a very tortuous ophthalmic vein, a sudden kink or constriction results, completely stopping the return of blood from the orbit to the cavernous sinus. As soon, however, as the venous pressure is lowered in the cavernous by a change of posture or relief from muscular strain, the kink or bend is relieved from pressure posteriorly, the vein emptied and the eye sinks back into its natural position in the socket. As there seems to be no record of an autopsy on a case of intermittent exophthalmos, our knowledge of this pathologic condition is largely theoretic.

In concluding this paper and especially from a study of the many artices comprising the index it is difficult to refrain from commenting on the gradual signs of progress being made year by year with reference to our better understanding of the anatomy, pathology and diagnosis of affection of the cavernous sinus.

TABLE 1

Septic Thrombosis of Cavernous Sinus

Doctor submitting information	Diagnosis	Etiology	Treatment	Result
Collins, A. N. Duluth, Minn.	Septic thrombosis	Purulent disease of nasal sinuses	Frontal sinus opened and drained	Death
Strout, E. Minneapolis, Minn.	Septic thrombosis	Accessory sinuses	Not given	Death
Hitz, H. B. Milwaukee, Wis.	Septic thrombosis	Not given	Not given	Death
Blair, V. P. St. Louis, Mo.	Septic thrombosis	Suppurative ethmoiditis	Accessory cavities drained	Death
Oppenheimer, S. New York City	(1) Septic thrombosis (2) Septic thrombosis	Aural Aural	Not given Not given	Death Death
Sudler, M. T. Lawrence, Kan.	Septic thrombosis	Aural	Not given	Death
Patton, J. M. Omaha, Neb.	Septic thrombosis	Boil of neck	Not given	Death
Geiger, Jacob St. Louis, Mo.	(1) Septic thrombosis (2) Septic thrombosis (3) Septic thrombosis	Chronic ear discharge Aural Aural	Not given Not given Not given	Death Death Death
Witherspoon, T. C. Butte, Mont.	Septic thrombosis	Typhoid fever—no details	Not given	Death
Lilienthal, H. New York City	(1) Septic thrombosis (2) Septic thrombosis (3) Septic thrombosis	Not given Osteomyelitis lower jaw Furuncle vestibule of nose	Exploratory craniotomy Not given Not given	Death Death Death

Rixford, E. San Francisco, Cal.	Septic thrombosis	Ascending infection pterygoid muscle and base of skull	Not given	Death
Gifford, H. Omaha, Neb.	Septic thrombosis	Aural	Not given	Death
Cott, G. F. Buffalo, N. Y.	Septic thrombosis	Aural	Not given	Death
Hamann, C. A. Cleveland, Ohio	(1) Septic thrombosis (2) Septic thrombosis	Peridental abscess Frontal and maxillary infection	Not given Not given	Death Death
Lynch, R. C. New Orleans, La.	(1) Septic thrombosis (2) Septic thrombosis	Aural Aural	Not given Not given	Death Death
Raymond, Alfred Seattle, Wash.	(1) Septic thrombosis (2) Septic thrombosis	Accessory sinuses Acute tonsilitis	Not given Not given	Death Death
Kyle, J. J. Los Angeles, Cal.	Septic thrombosis	Aural	Not given	Death
Jepson, Wm. Sioux City, .owa	Septic thrombosis	Accessory sinus disease	Not given	Death
Scholz, R. P. St. Louis, Mo.	Septic thrombosis	Aural	Radical mastoid and ligation lateral sinus	Recovery
Kanavel, A. B. Chicago, Ill.	Septic thrombosis	Neck infection	Local neck drainage	Recovery
Halstead, A. E. Chicago, Ill.	Septic thrombosis	Ethmoiditis and syphilis following ethmoid operation	Expectant treatment	Recovery

TABLE 2

Gun Shot and Penetrating Wounds of Cavernous Sinus

Doctor submitting information	Diagnosis	Treatment	Result
Babler, E. A. St. Louis, Mo.	1—Gun shot wound 2—Gun shot wound	No operation Not given	Death Death
Yates, J. L. Milwaukee, Wis.	Gun shot laceration		Death
Basham, D. W. Wichita, Kan.	Gun shot wound		Death
Raymond, Alfred Seattle, Wash.	1—Gun shot wound 2—Injury, right optic nerve and sinus torn across. Frontal lobe destroyed	Not given Tamponade	Death Recovery
Halstead, A. E. Chicago, Ill.	1—Gun shot wound through ear, lodging in sinus cavity. Developed sinus thrombosis and brain abscess. Sinus operated by Gasserian ganglion route and bullet removed. 2—Gun shot wound		Death Recovery, loss of both eyes

TABLE 3

Involvement From Fracture of Skull

Doctor submitting information	Diagnosis	Treatment	Result
Babler, E. A. St. Louis, Mo.	Fracture of base with rupture cavernous sinus	Not given	Death
Ochsner, A. J. Chicago, Ill.	Compound fracture with laceration cavernous sinus	Cavernous sinus tamponed	Recovery in ten days
Pearse, H. E. Kansas City, Mo.	Fracture base with laceration sinus wall	Operative removal of clot	Death—meningitis
Basham, D. W. Wichita, Kan.	Fracture with injury to cavernous sinus	Not given	Death
Bevan, A. D. Chicago, Ill.	1—Fracture of base causing cavernous sinus thrombosis 2—Fracture of base causing cavernous sinus thrombosis	Not given Not given	Death Recovery—loss of eye

TABLE 4

Rupture Internal Carotid Artery in Cavernous Sinus Arteriovenous Aneurism

Doctor submitting information	Diagnosis	Etiology	Treatment	Result
Schwyzer, A. St. Paul, Minn.	Pulsating exophthalmos—arteriovenous aneurism	Not given	Ligation common carotid	Cured now six years
Dennis, W. A. St. Paul, Minn.	Pulsating exophthalmos	Fracture base of skull	Ligation common carotid and ophthalmic vein	Relief, but not complete
Vaughan, G. T.	Arteriovenous aneurism	Not definitely ascertained	Ligation right internal carotid	Death
Martin, Edward Philadelphia, Pa.	1—Arteriovenous aneurism 2—Arteriovenous aneurism	Traumatic	Ligation orbital veins and carotid Not given	Relieved Not given
Lilienthal, H. New York City	Pulsating exophthalmos	Followed mastoid operation	Ligation both common carotids	Death
Griffith, J. D. Kansas City, Mo.	Arteriovenous aneurism	Not given	Ligation common carotid	Death
Dean, L. W. Iowa City, Iowa	Pulsating exophthalmos	Shot through orbit	Common carotid ligated	Improved
Bevan, A. D. Chicago, Ill.	Six cases aneurism internal carotid in cavernous sinus	Not given	Ligation common carotid	Recovery or very much improved
Halstead, A. E. Chicago, Ill.	Arteriovenous aneurism and thrombosis of cavernous sinus	Thrombosis followed ligation of carotid	Ligation internal carotid	Recovery from cavernous sinus thrombosis

TABLE 5

Injury to Cavernous Sinus During Miscellaneous Operations

Doctor submitting information	Diagnosis	Treatment	Result
Harris, M. L. Chicago, Ill.	During Gasserian ganglion operation	Tamponade controlling the profuse hemorrhage	Good; recovery
Davis, G. G. Philadelphia, Pa.	During Gasserian ganglion operation	Packing	Recovery
Halstead, A. E. Chicago, Ill.	During an hypophysis operation	Not given	Death from intracranial hemorrhage

TABLE 6

Malignant Growths Involving Cavernous Sinus

Doctor submitting information	Diagnosis	Treatment	Result
Benjamin, A. E. Minneapolis, Minn.	1—Sarcoma 2—Sarcoma	Details not given Details not given	Death Death

TABLE 7

Congenital Affections of Cavernous Sinus

Doctor submitting information	Diagnosis	Treatment	Result
Vanderveer, A. Albany, N. Y.	Congenital growth Fibrocystic	Resection zygoma and malar bone. Ligation of many veins surrounding cavernous	Good recovery

REFERENCES.

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XXIX.

A CASE OF VAGOTONIA, APPARENTLY ORIGINATING IN THE NASAL ACCESSORY SINUSES.*

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Vagotonia can be defined briefly as a condition of excitement or hypertonus of a group of nerves called the "extended vagus." The phrase "extended vagus" is applied to a nerve series which includes not only the pneumogastric, but also a group of nerves which functionate similarly to it. Antagonistic to the vagus group is the sympathetic, and there exists a condition of it called sympatheticotonia, which can be defined as a state of excitement or hypertonus of the sympathetic system. In order to locate accurately the "extended vagus" and sympathetic systems, a brief review of a few points in the anatomy of the nervous system might be in order.

Connected with the brain and spinal cord is a long series of paired nerves, beginning above with the first cranial and ending below with the last coccygeal. The fibers of these are divided into two groups, the animal and the vegetative. The animal group consists of sensory and motor fibers, and supplies the organs of sensation and the voluntary muscles.

The vegetative system supplies all those glands which have ducts, and all involuntary muscle tissue, including that of the muscular organs.

It has been found that this vegetative system consists of two distinct parts, called the autonomic system and the sympathetic system. These two cannot be separated by dissection, but through various tests, especially drug tests, their individuality has been established beyond question.

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The place of origin of the vegetative fibers is the brain and the spinal cord. The sympathetic portion arises from the cord alone, beginning at the second thoracic nerve and continuing down to and including the second lumbar.

The fibers which comprise the autonomic portion arise in part from the brain stem and in part from the sacral region of the cord.

The distribution of these fibers is widespread, but only that of the autonomic system will be given, as it is the one with which this contribution mainly is concerned. On account of direct clinical bearing, their distribution will be described in some detail.

The fibers which arise from the brain are contained in the third, seventh, ninth, tenth and eleventh cranial nerves.

Those of the third nerve go to the ciliary ganglion, whence other neurones take up the impulse and carry them to the ciliary muscle and the sphincter of the iris. It is probable, too, that the elevator of the upper eyelid receives autonomic fibers.

The fibers of the seventh nerve are those added to it by the pars intermedia of Wrisberg, and with those from the ninth, tenth and eleventh, go to sundry ganglia, whence secondary or postganglionic fibers emerge. These "innervate the blood vessels of the mucous membrane of the mouth, throat, nose, and paranasal sinuses, the salivary glands, the heart muscle, the glands and musculature of the trachea and bronchi, the glands and musculature of the digestive tube from the esophagus to the colon (gullet, stomach, intestine, liver and biliary passages, pancreas, and ducts), as well as the blood vessels and tubules of the kidney."[1]

The sacral part of the autonomic system derives its fibers from the first, second, and third sacral nerves, and is distributed through the pelvic plexus. It supplies fibers to the "musculature and glands of the lower colon, sigmoid, rectum, anus, urinary bladder, and genital organs, and to the arteries of the rectum, anus, and internal and external genitals."[1]

In action the two groups, the autonomic and the sympathetic, are antagonistic, and it is generally conceded that all glands which possess ducts and all involuntary musculature receive a supply from both. Upon the maintenance of a proper

balance between the two depends the normal functionating of the structures to which they go. If the autonomic supply is irritable, the organ will overfunctionate in one direction, and if the sympathetic is in a condition of hypertonus or excitement, the excess of action will be in the other. As glands and muscles are the organs to which these impulses go, the result will be manifested by hyper- or hyposecretion on the one hand, or by spasm or relaxation on the other.

The control of the entire vegetative systems, both autonomic and sympathetic, is believed to lie in the cerebrospinal axis, where possibly a regulating center exists, and in the glands of internal secretion, the socalled endocrinous glands. This latter is quite well established for the sympathetic system, adrenin having been shown to act generally as a stimulator to the entire distribution of this group. For the autonomic system no drug has been found which acts uniformly upon all parts of it. The nearest approach to such a drug is atropin, which is a sedative to practically the entire autonomic system, dilating the pupil, checking the flow of saliva and sweat, and relaxing contracted involuntary muscle. Eppinger and Hess[2] state that it is selective, in that the sacral part of the autonomic system is practically unaffected by it. In a recent paper, Block[3] takes an opposite view, as the result of having used atropin in what he calls the "vagotonic type" of dysmenorrhea, substituting this term for the older "essential or uterine dysmenorrhea." In this form of the disease he has used atropin in a limited number of cases with "most satisfactory results." Even more selective is pilocarpin, which has a powerful effect upon the salivary and sweat glands, producing oversecretion, and hence clearly to be regarded as a stimulator of part of this system.

Pilocarpin is known to be so reliable and constant a stimulator of the autonomic system that it is used in testing for the presence of vagotonia. One-twentieth to one-sixth grain is given hypodermically, and if the test is positive there will be noted salivation, lacrimation, sweating, cardiorespiratory arhythmia and hyperperistalsis, all these out of proportion to the size of the dose. Writers on this subject believe the drug tests to be quite important. The definition of vagotonia given by Eppinger and Hess[2] is as follows: "A functional, autonomic system disease, regarded so for the reason that all its

symptoms may be identified with those of a state of stimulation of the extended vagus or autonomic nervous system." But they do not consider this definition to be complete unless there is added to it "a hypersensitiveness to pilocarpin"[1] and "a relative decrease of reactivity to sympathetic stimuli."[2]

The symptoms of vagotonia in general are such as would be expected when the autonomic system is stimulated, and they represent activity of function at the terminal distribution of the nerves concerned. There are many of these symptoms, but just a few will be mentioned to indicate their general nature.

Cramp of the ciliary muscle, spasm of accommodation and widening of the palpebral fissure are examples of what would result from hypertonus of the fibers from the third nerve.

Salivation and congestion of and hypersecretion from the nose and nasopharynx would follow irritation transmitted through the otic, sphenopalatine and submaxillary ganglia.

To the vagus itself can be ascribed many phenomena, such as bronchial asthma, bronchial hypersecretion, laryngeal crises, bradycardia, gastric crises, hyperperistalsis and excessive gastric and intestinal secretion.

Through the sacral part of the autonomic system could pass impulses which would give rise to such conditions as spasm of the anal sphincter, hypersecretion from the intestine, dysmenorrhea, etc.

The blood picture shows an excess of eosinophiles. Hopkins[4] says: "Eosinophilia occurs especially where the skin, lung or intestinal element is present, and is increased after pilocarpin, often markedly."

An interesting observation by Eppinger and Hess[3] is that in vagotonics there is likely to be a marked anesthesia of the entrance to the pharynx, a condition usually ascribed to hysteria. These authors say: "We wish to state that, on the basis of our observations, we regard this phenomenon as entirely unrelated to hysteria."

The treatment of the condition is the same as that of any other secondary disease, viz., find, if possible, and eliminate a cause or focus, build up the health and give proper medication. The last factor, the medicinal treatment, can be managed on one of two principles, either administering a sedative

for the autonomic system, or overcoming the tonus of the latter by giving a stimulator to its antagonist, the sympathetic system. This principle is illustrated notably by the successful treatment of spasmodic bronchial asthma, success being possible either by quieting the autonomic system with atropin or by stimulating the sympathetic by the local or hypodermic use of adrenin.

The case I wish to report in this connection was referred to me by Dr. Alfred Stengel, who had given it long and careful study. The patient was a lad of about twelve years, who for five years had gradually failed in health, with an intermission or two of slight improvement. He presented many symptoms of autonomic tonus and, in addition, had evident disease of his paranasal sinuses.

His impaired health dated back to an attack of measles at the age of seven, which left him with marked nasal discharge and expectoration, which have persisted ever since. There was at times a loose cough, which was productive of a thin, mucoid sputum, which never became yellow nor blood tinged. For a year there was profuse sweating at night, and his sleep was much disturbed by sneezing, coughing and expectorating. His adenoids were removed during this time, but the result was unfortunate, as immediately afterward his nasal discharge increased and he developed abscesses in both ears.

Three years after the appearance of the first symptoms Dr. Stengel saw him. No cause for his symptoms was found, physical examination being negative. He was tested with pilocarpin for vagotonia by Dr. Hopkins, and the result was positive. He was put upon guaiacol carbonate and extract of belladonna, and some slight improvement resulted.

Two years later he returned to Dr. Stengel, a very sick little boy, having lost nine pounds in four weeks. At this time he was vomiting all his meals, a condition which soon changed to a weekly attack of severe abdominal pain, nausea and vomiting, which would last about two days. The vomitus was profuse and consisted almost entirely of mucus. With this there was profuse salivation, of a clear, watery character. Soon was added to these conditions frequent watery bowel movements. Medication was of little value, and the boy's condition was becoming a desperate one.

It was at this time I was requested to examine him, and aided by an X-ray examination by Dr. H. K. Pancoast, a diagnosis of chronic suppuration in the posterior ethmoidal and sphenoidal cells of the left side readily was made. The surgical indications were clear, and I removed the posterior end of the middle turbinated and opened widely the offending cells.

Improvement was immediate and very striking, as almost at once his symptoms disappeared. In two months he gained ten pounds and was a perfectly well boy except for some discharge from his nose. Later Dr. Herbert Fox administered autogenous vaccines, and this was followed by almost total disappearance of the nasal mucus.

Our interpretation of the case is this: The boy was one with a very unstable vegetative nervous system, and it has been observed that in such a state small stimuli can produce large reactions. The irritation which would cause such a condition of autonomic hypertonus may (quoting Hopkins[4]) "arise from noxa in the form of bacterial toxins, as during or after acute infections, from drugs or from the products of deranged metabolism, mechanical irritation and so forth." There was present in this case a primary acute infection, measles, followed by prolonged chronic suppurative sinus disease, and we believe that the consequent toxemia and irritation were the exciting factors. For, when this was relieved, the widespread vagotonic symptoms disappeared so promptly as practically to bar accidental coincidence.

As far as I have been able to discover, this case is the first to be placed on record in which such a distinct entity as chronic sinus disease was presumably the exciting cause of such widespread vagotonic manifestations, and in which prompt disappearance of these symptoms followed relief of the local condition. By the free drainage of suppurating sinuses this patient was almost instantaneously relieved of salivation, cough, expectoration, severe abdominal pain, vomiting and diarrhea, for none of which symptoms a physical cause could be found.

As a sinus case per se there was nothing notable about it, as the diagnosis was easy and the treatment simple. The big feature is the field of observation and deduction such a case opens up. It does seem that many other conditions which are but vaguely understood may possibly become clarified when

viewed as vagotonic or sympatheticotonic phenomena. For example:

The treatment of bronchial asthma with adrenin or with atropin has been successful in many instances.

Noteworthy has been the nasal treatment by our colleague Emil Mayer,[5] Brettauer[6] and others, of selected cases of dysmenorrhea.

Another fellow member, Greenfield Sluder,[7] after painstaking study, has succeeded in relieving certain symptoms by treating the sphenopalatine ganglion.

These are but a few of the conditions upon which some light may be shed. Undoubtedly many others will suggest themselves as being based upon a derangement of visceral nerve supply. Should such prove to be the case, they will constitute a new group whose pathology can be described in definite terms.

In conclusion. An obvious criticism which may be and has been made of this entire subject is that there is much of the speculative in it. In answer to this comment, Barker[1] says: "We find out new facts by forming hypotheses. No man works without an hypothesis. So long as we use hypotheses only to stimulate work, to clarify our views, or to hold even temporarily widely separated facts together, I think they may be most helpful."

Eppinger and Hess,[2] with the same thought in mind, say: "We know full well that many of the things which we have sated stand but on unsteady ground, but, in spite of this, we hope to have given the impetus to further investigations which may be of great significance for special pathology and therapy."

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XXX.

A CASE OF SEPTIC THROMBOSIS OF THE JUGULAR BULB, WITH REPEATED FORMATION OF SEPTIC THROMBI IN THE SIGMOID AND LATERAL SINUSES. WITH REFERENCE TO THE LITERATURE ON INVOLVEMENT OF THE TORCULAR HEROPHILI IN SUCH CASES.*

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The proximity to the torcular Herophili of the final thrombus which formed in this case, and the cause of its limitation at this point, have brought to mind some conflicting views regarding the tendency of a thrombus in this region to extend across the torcular to the opposite side. The discussion of this subject by Mr. Ballance and Mr. H. E. Jones, senior surgeon of the Liverpool Eye and Ear Infirmary, will be referred to later.

A delicate girl, twelve years of age, developed an abscess in her right middle ear, upon which a myringotomy was performed the following day. The third day after the myringotomy was performed she had a chill, and her temperature rose to 103.8° by mouth.

The following day I saw her and advised immediate mastoidectomy. Nothing unusual was found beyond a well infected mastoid, but because of the chill on the previous day and the septic appearance of the child, a guarded prognosis was given, and the precaution was taken to remove the plate over the vertical limb of the sinus for inspection of its wall. It was apparently normal, but the possibility of infection being in the bulb was remarked at the time.

The patient, being operated on out of town, was left in the care of Dr. Kellogg, of Norwalk, Connecticut, who reported

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the following morning that the temperature had gone to 106.5° during the night, but that the condition was then good, temperature at noon being 99.8°. In the afternoon, after a chill, the temperature rose to 107.5°.

Exploration of the sigmoid sinus that night revealed scant bleeding from the bulb, but no clot in the vein, and no apparent change in its wall. The internal jugular was resected and the lower portion of the sigmoid sinus wall cut away.

A fall in the temperature to 98°, accompanied by an alarming collapse, followed three hours after the jugular resection. Next day, however, the child's condition was fair and spirits good, but the following night the temperature rose to 107.2°, and the next night to 105.5°. After this her condition improved, but at the end of a week signs of sepsis began to return.

On the twelfth day after the jugular resection a large abscess was opened in the upper part of the vertical limb of the sinus. The sinus wall was excised beyond the clot, and free bleeding was obtained. Culture from the pus showed streptococcus hemolyticus; blood culture was negative—leucocytes 20,000, ninety-two per cent polynuclears. She now complained of blurred vision. A distinct double optic neuritis was present, more marked on the right side. The disturbance of vision apparently subsided in a few days, and no further ophthalmoscopic examinations were made.

Immediate improvement followed this operation, but within a week a return of the high temperature and sepsis made further exploration of the sinus in the direction of the torcular necessary.

Because of the patient's extreme weakness the operation had to be performed in bed without an anesthetic. More bone was removed and the sinus further exposed for about two centimeters. Incision of it released another dram of thick gray pus from its interior. Gentle removal of the torcular end of the thrombus was made with forceps, and a good flow of blood was obtained which flushed out the vein. Iodoform gauze was then introduced into the lumen of the vein for one inch or more, with the hope that the clot might be induced to form beyond the area of infected wall. This was followed by marked improvement for six days. On the seventh day signs

of sepsis reappeared, and a fourth exploration of the sinus still further back was necessary.

At this operation (under ether) an abscess, similar to the other two, was found, the sinus being filled with dark gray pus, which extended back to within three centimeters of the posterior occipital protuberance. There were no granulations on the wall, but it had an infected appearance. The clot in the vein, which extended beyond this point, was removed with forceps, and the bleeding which followed its removal was controlled by my finger in the vein. With my finger in the vessel for two or more centimeters I could feel a septum which seemed to divide its lumen at the torcular.

Feeling that the vein wall might be infected to this point, I inserted iodoform gauze as far back as my finger reached, and later felt fearful lest it might produce a thrombus in the other sinuses. This gauze was removed on the second day, and its removal was followed by a surprisingly small flow of blood, due probably to a partial thrombosis of the superior longitudinal and a naturally small communication with the sinus of the opposite side.

During the progress of the case, which after this operation made an uninterrupted recovery, Dr. John D. Richards expressed the opinion that the thrombus would not extend to the opposite sinuses of the torcular, as the rapid flow of blood through them at this point would tend to limit the formation of the clot.

In this opinion he is supported by Mr. Ballance,[1] who, in 1890, made the following statement: "It is an error to suppose that the lateral sinuses are continuous at the torcular Herophili; the right sinus is continuous with the superior longitudinal sinus, and the left sinus is, as a rule, a continuation of the straight. There are sharp bends in the venous channels where the lateral sinus commences, and these are joined across the front of the internal occipital protuberance by means of a separate sinus, or cross branch, of small size; thus a thrombosis of one lateral sinus need not, and probably will not, be accompanied by thrombosis of the other lateral sinus, and so life is spared." While the above arrangement is not invariable, it will be found to be the general rule.

Cunningham, states[2] that "the superior sagittal sinus turns

to the right and becomes continuous with the right transverse sinus, while the posterior end of the straight sinus turns to the left and joins the left transverse sinus. In a certain number of cases this arrangement is reversed, and not uncommonly there is a communication between the right and left transverse sinus across the front of the internal occipital protuberance." He then says that "occasionally the superior sagittal, the two transverse sinuses, the straight sinus, and the occipital sinus unite anterior to the occipital protuberance in a common dilatation—the 'confluens sinuum.'"

Despite the fact that this last arrangement is the most infrequent of any, it seems, from the descriptions given of operations on the transverse sinus in this region, the one thought to be most common. This very general impression has been due to the meager descriptions of the textbooks on anatomy, which refer to the torcular Herophili only as a marked dilatation of "irregular form" where the five sinuses meet.

In regard to the complete separation of the two sides Dwight[3] says, in a discussion of the subject, that he "cannot agree with Rüdinger, that complete separation of the two parts of the torcular is common, this and perfect symmetry being about equally rare." The right lateral sinus is usually considerably larger than the left, and the asymmetry may extend to complete absence of one lateral sinus and jugular vein, as has been referred to by Eagleton[4] and others.

Hugh E. Jones,[5] senior surgeon of the Liverpool Eye and Ear Infirmary, is of the opinion, contrary to Mr. Ballance, that "once at the torcular the progress of infection to the other great sinuses is easy," and that the question of extension of infection to the opposite sinus has not received the attention it deserves in literature. He supports this opinion by reference to a case reported by himself, a remarkable one by Whitehead,[6] and one by Lodge; but with the exception of Whitehead's case, the others appear to me not to have been due to gradual extension of the thrombus from one sinus to that of the opposite side, but to the formation of a clot shortly before death in the whole region of the torcular, due, in all probability, to the general condition of the patient rather than to a true extension of the septic thrombus from the lateral sinus; for while thrombosis of the whole region of the torcular and all the sinuses has been repeatedly reported in cases profound-

ly septic and debilitated from various causes, so far as I have been able to ascertain the literature records few cases in which a truly septic thrombus extended from the mastoid region of one side across the torcular to the opposite side.

The extension would naturally take place only to that sinus of which the one involved was a continuation, and because of the usual separation, except for the rather variable communication between the two sides, it would not be likely to extend to the other side.

An example of this is prettily demonstrated in a case reported by Dr. Simpson,[7] in which the mastoid and lateral sinus of the left side had been operated on. Autopsy showed "the superior and inferior longitudinal sinus to be patent and without change, the left lateral sinus completely occluded, a thrombus extending from the point of operation at the left mastoid to the torcular Herophili and also to a slight extent into the straight sinus. The right lateral sinus was quite free and healthy."

In the case referred to by Mr. Jones[8] the autopsy report says: "From the junction of the superior petrosal sinus with the lateral sinus up to within one-half inch of the torcular Herophili, the walls of the sinus were healthy, but here commenced a patch of phlebitis one inch long, etc., etc, which extended across the midline to the superior longitudinal sinus and right lateral sinus." It will be seen from this that there was not an extension from the original thrombus in the sigmoid, but that one formed spontaneously in the torcular, due probably to the extremely septic and weakened condition of the patient.

In another article,[8] in opposition to Mr. Ballance, he cites the twenty-two cases of lateral sinus thrombosis associated with ear diseases, collected by Newton Pitt,[9] in which the clot extended to the longitudinal sinus in four, to the opposite lateral in three, and in one to all the sinuses, and says that "not only, therefore, is it a not unlikely occurrence, but a comparatively common one." Again we think his conclusion unjustified, for it will be seen from this collection of Pitt's that in four cases the clot extended to the longitudinal sinus (a continuation of the right lateral), and not to the opposite side; so while this leaves four to be accounted for, it cuts down his percentage by half. And among these we are not convinced

that there were not some similar to the one he reported in which the thrombosis was due to general causes rather than to a real extension from the primary thrombus in the mastoid region.

If I may be permitted to cite one more reference, Lidell,[10] in 1874, collected thirteen cases of sinus thrombosis associated with mastoiditis in which autopsies were performed, and in one of these there was extension a short distance into the opposite side. The report says: "A light colored, purulent looking plug distended the right lateral sinus and adhered closely to its walls—the same clot filled the torcular Herophili also, and extended a little way into the left lateral and straight sinuses. The posterior two-thirds of the superior longitudinal sinus was filled with greenish pus, which also extended to the veins coming from the pia on the right side."

This report, to my mind, supports Mr. Ballance rather than Mr. Jones, for the superior longitudinal sinus was, no doubt, continuous with the right lateral, in which the trouble began, and two-thirds of its length became involved before the torcular was crossed and the opposite lateral and straight sinus were involved, even for "a little way."

In Whitehead's very exceptional case, which recovered, there was no doubt of the involvement of both sides of the torcular.

After operation on the right mastoid he says: "The sinus was freely exposed and found to be thrombosed; the overlying bone was removed back to and over the torcular Herophili and the sinus laid open the whole distance; the openings of the longitudinal sinus and of the lateral sinus of the other side were blocked with firm clot, and a small curette passed into the opposite sinus failed to set up bleeding." Such cases are very rare.

While we not infrequently hear of a thrombus being encountered which extends from the torcular to the bulb, and occasionally a case in which no bleeding is obtained from the torcular region, no minute descriptions are given of the operations in this region, and I think it will prove interesting to observe as often as possible in the living, and at autopsies, just what direction and to what extent lateral sinus thrombosis progresses in this region. It will be interesting also to note more carefully in future the variations in the anatomy of this interesting meeting place of the sinuses.

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XXXI.

MALARIA SIMULATING DISEASES OF THE MASTOID AND FRONTAL SINUS.*

By Charles W. Richardson, M. D.,

Washington.

Malaria, like syphilis, affects many organs and tissues of the body, producing lesions thereby, and through action upon the nervous system may simulate diseases of other organs and tissues which are not directly affected by the parasites of these diseases.

I wish to present to you today two interesting cases wherein the malarial parasite, through its invasion of the organism, produced striking subjective evidences in one case of mastoiditis, and in the other of frontal sinuitis. Beyond these interesting facts, these cases present nothing of unusual interest. The diagnoses were readily made through exclusion, without any difficulty, and were confirmed by the blood examinations.

The first case, that of simulating mastoiditis, was brought to my attention by my assistant, Dr. W. H. Huntington, immediately after I had finished a rather laborious afternoon in the operating room. The provisional diagnosis was mastoiditis, with probably intracranial complications. Due to lack of time, I made only a cursory examination, and quickly reviewing the history, found the evidence such as to warrant a strong doubt in my mind both as to the mastoid infection as well as the more serious phase of the case. I ordered a blood examination and count and urinalysis. Dr. Huntington had already made an examination of the eye grounds. In the late afternoon I made a most thorough examination of the patient, and through the history and the results of this examination I became convinced that the mastoid symptoms were due to other causes than sup-

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puration. At this time I had also the benefit of both the blood examination and the urinalysis.

The history of the case, as prepared by Dr. Huntington, is as follows:

Case 1.—Miss M. L. H., female, white, twenty-one years of age, was admitted to the Episcopal Eye, Ear and Throat Hospital, December 8, 1915. Family history negative.

Previous History.—Had measles when eight years of age. Had chills during infancy. No previous affection of the ears.

Present Illness.—Marked evidence of great prostration, requiring support in walking. Very pale, Had acute suppurative otitis August 15, 1915, with spontaneous rupture of membrana tympani. Discharge continued until November 6, 1915, when it ceased. At this time postauricular pain became manifest and gradually grew worse, with swelling back of ear and in neck. About a week later headaches, which were not constant and confined to occipital region. Vomited at times. About second week in November had chills, which were unattended with sweats. Complained of great thirst and drank abundance of water. Urine at that time was scanty and very dark. During past few days has had marked pain over mastoid region. For past two weeks has noticed gradual diminution of vision.

Physical Examination.—Left ear normal. Right ear shows a central kidney-shape perforation. Ear dry. Pressure over mastoid, as well as along course of jugular vein, causes pain. No edema. Pulse 120. Temperature 101°.

Eye.—Slight exophthalmos. Pupils widely dilated, but reacted to light and accommodation. No nystagmus. Vision impaired. Figures at ten feet. Eye ground shows bilateral papillitis, with hemorrhage and exudate around vessels. Arteries small. Veins congested and tortuous. No exudate at macular region.

When I first saw this patient I was impressed with her hysteric facial expressions and marked pallor. Examination of ears demonstrated them to be as given in the history. The tenderness over the right mastoid was very great, as well as along the course of the jugular vein. When the patient was placed upon her feet and told to walk, she would advance a few steps and then begin to totter, showing rather an evidence

of weakness than of any central lesion. The double optic papillitis also excited my suspicion, as there were no evidences of any acute cerebral lesion that could account for its centric origin. The head pain, the pallor and the slight mental hebetube inclined me to await an examination of the urine and blood before making a diagnosis.

The urinalysis made from urine drawn by catheter was as follows: Color, dark brown, very turbid. Specific gravity 1023. Reaction, acid. Sugar, considerable quantity. Albumin, trace. Microscopic: Casts, granular, hyaline and epithelial, with a predominance of granular. Many disintegrated blood cells.

Blood Examination.—Malarial parasites present. Leucocytes, 8,800.

The patient was given quinin in large doses, large draughts of water, the Murphy drip and hot packs. Under this treatment the temperature gradually subsided from 101.2° on entrance to 99°. On December 11th there was a rise in temperature to 102°, unattended with chill, which subsided to normal December 12th. From December 12th until the patient was discharged the temperature was flat. Blood examination, made after December 12th, demonstrated a freedom from malaria. The urinalysis showed normal urine after December 16th. The mastoid tenderness and the general condition of the patient improved from day to day. The papillitis improved considerably, and the vision became clearer, but neither had returned to the normal when the patient was discharged from my service.

January 4, 1916, some weeks later, the patient returned to the hospital on account of the want of further improvement of the eye condition. On account of a brother of the patient, an attendant in the dispenary service, having been found to be a syphilitic, a Wassermann was made upon the patient with a positive finding.

Case 2.—The case of frontal sinuitis presents a more clearly defined history of malarial origin than the case of mastoiditis, as it was uncomplicated. The patient was a young locomotive fireman, who lived in a malarial district in the State of Maryland. When I first examined him he manifested all the subjective evidences of a frontal sinus infection. He complained

of intense frontal head pain. There was a marked tenderness over the left frontal region, which was particularly marked in the floor of the sinus. Inspection of the nose demonstrated an absolutely normal nasal chamber. Postnasal examination also showed no deviation from the normal. The following day I again went carefully over the patient, and from the total absence of all objective evidences of frontal sinus infection, and from the fact that the patient had dwelt in a malarial district, I directed that a blood examination and count be made. The patient was admitted to the Episcopal Eye, Ear and Throat Hospital, December 28, 1915. The history is as follows:

J. W. S., twenty-four years of age, living in Seat Pleasant Heights, Maryland. Mother, living, well; age, forty-seven years. Father, living, well; age, fifty-one years. Maternal grandmother, living, well; age, seventy-four years. Paternal grandmother, living, well; age, seventy-five years.

Diseases Previous.—Measles when two years old; chicken pox when four; whooping cough when ten; malarial fever, eight years.

Physical Examination.—Weight, 160 pounds. Height, 5 feet, 10½ inches. Chest measurement 36 inches expanded, 40 inches insp. Waist measurement, 33 inches; robust, well formed. Heart, lungs, liver, joints, etc., normal. Tenderness and pain over left eye increased on pressure.

Personal Habits.—Lives in a swampy, poorly drained district; is employed as a locomotive fireman. Uses tobacco moderately. Uses alcohol moderately, a glass of beer or whiskey each day, sometimes two; more if on party. Retires at 10 p. m., rises 5 a. m. Sleeps well at night in house not well screened. In the summer time is in the habit of sitting on unscreened porch, and is bothered a great deal by the mosquitoes. Has not been away from home in eight years. Is a big meat eater twice a day, averaging one and a half pounds of meat per day.

Present Illness.—About five weeks ago patient had a heavy cold on his chest, later extending into the nose and pharynx (naso-). This sickness began with a severe rigor, accompanied by pains in the head, particularly at the frontal region. The patient also complained of a bitter taste in the mouth. Had a temperature for the first week, pains in the back and legs,

malaise, no appetite, bowels regular, frequent micturition. The pains in his head increased, and Dr. Apgar sent him to the Episcopal Eye, Ear and Throat Hospital. Upon admission he was suffering from pain and tenderness over the left eye and the frontal region of the forehead. Pain was markedly increased upon pressure. No discharge from the nose other than normal. Pain was not increased by the reclining posture.

Clinical Examination.—Urine showed nothing abnormal. Blood showed the presence of a large number of tertian malarial parasites. Scarcely one erythrocyte could be found free from the parasite. The leucocytes were at first decreased, five thousand per cubic millimeter; later there was a leucocytosis, twelve thousand per cubic millimeter.

Treatment—Quinin sulphate, grains five every three hours, was given for the first four days. The malarial parasites showed marked decrease in numbers. The fifth day they showed increasing numbers. The quinin was then increased to grains five every two hours. The parasites again decreased and almost entirely disappeared. The pain over the left eye and frontal region entirely cleared up, and the patient was discharged as cured of the condition for which he came to this hospital.

Diagnosis.—Malaria involving the frontal sinus.

Prognosis.—Favorable.

It is interesting to note that although the patient was infected through a tertian malarial parasite, he had never had a chill, fever, or other evidences of malarial infection. During the two weeks that he was under our observation he ran a normal temperature. I never saw a field in a slide from a malarial patient that showed more parasites in it than did the slide which I examined of this blood. Under appropriate antimalarial treatment the parasites disappeared from the blood. With the disappearance of the malarial infection the frontal sinus pains and tenderness also gradually passed away. The patient was discharged cured at the end of two weeks.

XXXII.

THE INNERVATION OF THE NASAL CHAMBERS.*

By Albert P. Brubaker, M. D.,

Philadelphia.

It was a pleasure and an honor to have been invited by the program committee to present to you, though briefly, some of the anatomic and physiologic facts relating to the innervation of the nasal chambers and their associated structures.

The secretomotor and vasomotor nerve mechanisms that excite and regulate the secretory discharge and the vascular supply of this region, and the manner in which they in turn are excited, not only to normal but to abnormal activity, seemed an appropriate subject for presentation and perhaps discussion at a meeting of this character. In the short time at my disposal, only the more essential facts regarding these mechanisms, in so far as they have been described, will be alluded to.

The mucous membrane lining the nasal chambers is thick and spongy and provided with a large number of glands, some of which secrete a fluid rich in mucin, while others secrete a fluid containing albumin, and hence fall into the two categories of mucous and serous glands. The surface of the mucous membrane is covered for the most part with tall columnar cells, between which are to be found mucus or goblet-shaped cells, so called from the appearance they present after the discharge of their contained masses of mucin. The blood vessels distributed to the mucous membrane, and which supply the glands and epithelial cells with the raw materials for the elaboration of their secretions, are mainly branches of the sphenopalatine arteries—subdivisions of the internal maxillary—though branches of the facial are also contributory. Veins which correspond in a general way to the arteries return the venous blood to the general circulation.

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In order that the glands may be enabled to elaborate and discharge the necessary amount of secretion, it is essential that the vascular supply be varied from time to time in accordance with the nutritional needs and functional activities of the mucous membrane and its contained glands. The secretion of the glands as well as the blood supply no doubt impart to the nasal chambers that degree of moisture and temperature necessary to prepare the inspired air in both respects for its entrance into the lower respiratory organs.

Though living, and therefore possessing the fundamental property of all living material—viz., irritability—the epithelium lining these glands, as well as the muscle cells in the walls of the arterioles, are lacking in spontaneity of action, and therefore require for the manifestation of their activities the stimulating influence of nerve impulses originating in and transmitted from the central nervous system by specialized groups of nerve fibers. The nerves distributed to these two structures belong to what is termed at the present time the autonomic system, and hence consist of two consecutively arranged neurons.

THE EFFERENT NERVE MECHANISM.

The origin, course and distribution of the secretomotor and vasomotor nerves for the glands and blood vessels of the nasal mucous membrane have been fairly clearly determined in recent years. The results of many lines of investigation are represented in the accompanying diagram.

In a description of this mechanism it will be convenient to consider, first, the postganglionic fibers and their distribution; and second, the preganglionic fibers by which they are associated with the central nervous system. The postganglionic fibers that directly excite to activity the epithelium of the glands and the muscle fibers of the blood vessels have their origin in the nerve cells which constitute the sphenopalatine, or Meckel's ganglion, situated in the sphenomaxillary fossa. From this origin the nerve fibers pass in different directions to become ultimately related, histologically and physiologically, with the epithelium of the glands and blood vessels, not only of the nasal chambers, but of the roof of the mouth, palate and upper portion of the pharynx as well. The stimuli that call

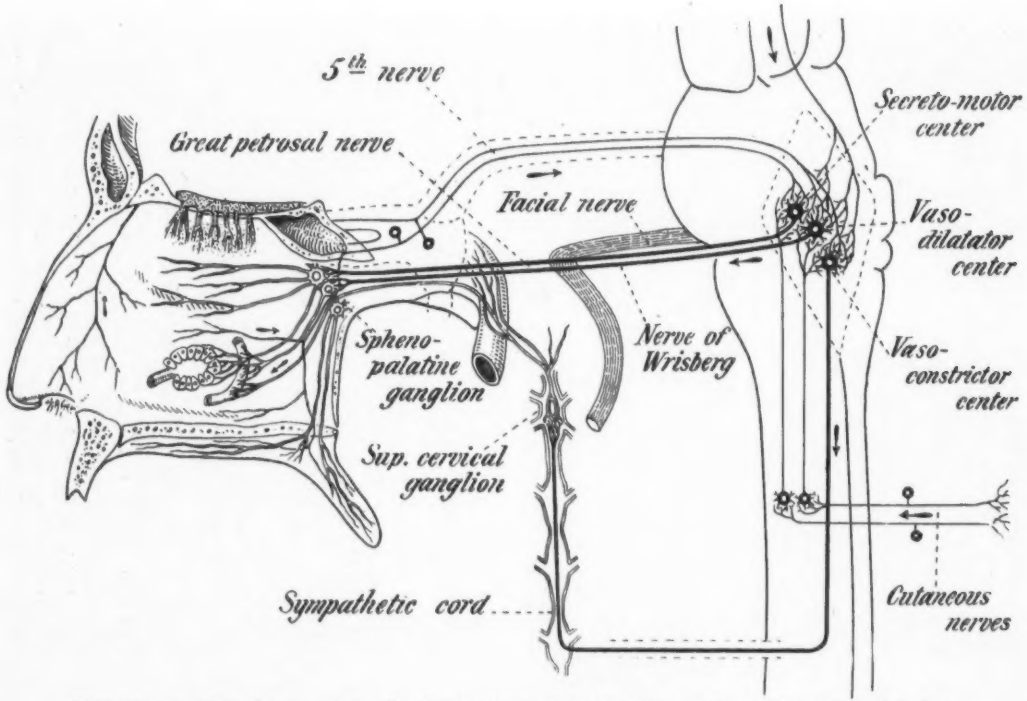

Diagram showing the nerve mechanism concerned in the innervation of the nasal chambers.

forth the activities of these peripheral structures are nerve impulses discharged by the ganglion cells and transmitted to them by the cell processes and their branches. But the ganglion cells, though irritable, are also devoid of spontaneity of action, and require for their excitation the arrival of nerve impulses, discharged by the nerve cells in the central nervous system and transmitted by the preganglionic fibers.

The preganglionic fibers have their origin in nerve cells situated in the gray matter beneath the floor of the fourth ventricle. From this origin the nerve fibers pass forward and emerge from the side of the medulla, in a small bundle, between the facial and acoustic nerves, known as the pars intermedia, or nerve of Wrisberg, of which they constitute a part. Just after these nerves in their forward course enter the internal auditory canal, the nerve of Wrisberg enters the sheath of the facial, having but a short independent course. At the point where the facial nerve turns backward to enter the aqueduct of Fallopius, some of the fibers in the nerve of Wrisberg leave the facial and pass forward to the sphenopalatine ganglion, around the cells of which the terminal branches of the nerves arborize, thus forming histologic and physiologic connections with them. These fibers in their course from the facial to the ganglion are termed in works on anatomy the great petrosal nerve.

The central cells, situated in the ventricular gray, are thus brought into relation directly by their preganglionic fibers, with the ganglion cells, and indirectly, by the postganglionic fibers, with the glands and blood vessels. If, therefore, nerve impulses were to be discharged by the central cells, they would be transmitted by the nerve fibers of the nerve of Wrisberg and their continuations—the great petrosal nerve—to the cells of the sphenopalatine ganglion, which would then become aroused to action; these in turn would then discharge impulses which, owing to the extraordinary branchings of their processes, would be distributed in a spray-like manner to the myriads of epithelial cells lining the glands and to the myriads of muscle cells found in the walls of the arterioles.

That this is the pathway for the transmission of nerve impulses from the central cells to the foregoing tissues is shown by the results of experiment. Thus, if the great petrosal nerve

is exposed in one of the higher mammals and divided, and the peripheral end stimulated with induced electric currents, there will arise a dilatation of the blood vessels and a discharge of secretion from the glands of the mucous membrane of the nose and associated structures. Stimulation of the sphenopalatine ganglion or its branches gives rise to a similar series of phenomena. An experiment of this character discloses the fact that this nerve contains both secretomotor and vasodilatator fibers, and the further fact that the central cells from which they arise are divisible into two groups—viz., secretomotor and vasodilatator. There is no evidence that the great petrosal nerve contains any fibers from the facial proper for the innervation of any striated muscle of the palate.

The blood vessels of this region, however, are regulated in the degree of their contraction partly by vasoconstrictor nerves. These nerves also consist of two consecutively arranged neurons and, therefore, belong to the autonomic system. The peripheral one arises in the superior cervical ganglion of the vertebral chain. From this origin the nerve processes—postganglionic fibers—pass upward and assist in the formation of the plexus surrounding the internal carotid artery. At the level at which the great petrosal nerve crosses the artery, some of these fibers enter the sheath of the great petrosal and accompany its branches to their terminations—viz., the walls of the blood vessels and possibly the cells of the glands as well. The preganglionic fibers have their origin in cells forming a part of the general vasoconstrictor center, situated in the gray matter beneath the floor of the fourth ventricle. From this origin the fibers descend the spinal cord as far as the thoracic region, where they pass out from the cord in the ventral roots of the upper thoracic nerves. After a short distance they leave the ventral roots and enter the vertebral chain of ganglia and pass upward to the superior cervical ganglion, around the cells of which their terminal branches arborize. There is thus established a rather circuitous pathway between the vasoconstrictor center and the blood vessels of the nasal mucous membrane and the associated structures. That this is the pathway for the transmission of nerve impulses from the vasoconstrictor center to the blood vessels is shown by experiment. Thus, if the cervical sympathetic cord

is divided, not only the blood vessels of the nasal chambers, but of the entire side of the head and face, will dilate and become filled with an increased volume of blood, a condition which will last for some days. If now the peripheral end of the divided cord is stimulated with induced electric currents, the blood vessels at once contract and remain contracted as long as the stimulation is continued.

It would thus appear that the blood supply is under the control of two antagonistic groups of nerves, a dilatator and a constrictor group. The caliber of the blood vessels at any one moment is the resultant of the relative degree of activity of these two groups. According as the one or the other predominates will the caliber of the vessels be increased or decreased, and the blood supply increased or decreased in the same ratio.

THE CENTRAL MECHANISM.

The central mechanism for the innervation of the nasal chambers is thus shown to consist of groups of nerve cells having secretomotor, vasodilatator or inhibitor, and vasoconstrictor or augmentor functions.

The question now arises as to the manner in which these different groups are excited and coordinated in action. Experimental work has demonstrated that as a primary condition for their activity they must possess a certain degree of tonicity or tonus; a degree of excitation due to a reaction between the constituents of the cells and the constituents of the lymph and blood by which they are surrounded. Given this tonus they may be excited to activity or inhibited by:

1. The arrival of nerve impulses transmitted from the mucous surfaces of the nasal chambers by the nasal and other branches of the trigeminal nerve, and from the skin by the cutaneous nerves.

2. The arrival of nerve impulses descending from the cerebrum in consequence of psychic states of an affective or emotional character.

In the former instance the excitation is said to be reflex or peripheral in origin; in the second instance, direct or cerebral in origin. In either instance the resulting change in secretion and vascularity will be proportional to the intensity of the peripheral or the cerebral excitation and the degree of tonus

possessed by the central cells themselves. It is evident from a study of pathologic conditions that the tonus of one or more of these centers may be abnormally increased or decreased by changes in their nutrition; changes due to alterations in the composition of the lymph and blood, the result of increased production or diminished elimination of the more or less toxic products of metabolism. It is a widespread observation that the perverted functions of the nasal chambers are closely associated with dietetic or alcoholic excesses, in conjunction with an insufficient amount of physical exercise and fresh air. With the establishment of an impaired tonus of the central mechanism, peripheral causes which at one time would produce slight if any effect, may now give rise to the most pronounced and distressing effects.

THE AFFERENT NERVE MECHANISM.

The afferent portion of this nerve mechanism is to be found in the branches of the trigeminal nerve. The fibers composing the sensor portion of this nerve have their origin in monaxonic cells in the ganglion of Gasser. (See diagram.) The cells of this ganglion give origin to a short process which soon divides into two branches, one of which passes centrally, the other peripherally. The centrally directed branches collectively form the socalled large or sensor root; the peripherally coursing branches collectively constitute the three main divisions of the nerve—viz., the ophthalmic, the superior maxillary, and the inferior maxillary.

The axons of the centrally coursing branches, after entering the pons Varolii, ultimately aborize around the vasoinhibitor, the vasoconstrictor and the secretomotor centers; the peripherally coursing branches pass forward to be distributed to the skin, mucous membrane and other structures of the head and face. By this means the nasal mucous membrane is brought into anatomic and physiologic relation with the foregoing nerve centers. Other of the centrally coursing branches aborize around other groups of nerve cells, the socalled "sensor end nuclei," the axons of which pass upward to and aborize around sensor nerve cells of the cerebral cortex. Stimulation of the first group calls forth reflex phenomena; stimulation of the latter group evokes conscious sensations of a painful character.

The cutaneous nerves associated with this mechanism have a similar origin and distribution.*

REFLEX OR PERIPHERAL EXCITATION OF THE CENTRAL MECHANISM.

The stimulus to the peripheral terminations of the nasal and cutaneous nerves is the variation in the temperature and moisture of the atmospheric air.

If one of the functions of the nasal chambers is the warming and moistening of the inspired air to that degree best adapted for its entrance into the lower respiratory passages, it is apparent that the amount of heat radiated from the blood and the amount of secretion produced by the glands will, or ought to, vary with the variations in the temperature and moisture of the inspired air. Since these latter factors may vary simultaneously in the same or opposite directions, it is apparent that the former factors will, or ought to, vary in accordance with them in the opposite direction.

The phenomena that will be observed are difficult to predict, for the reason that the reactive adaptations of individuals vary with a variety of factors—age, sex, occupation, habits of life, stability of the nerve centers, etc. However, in a general way it may be stated that if a fairly normal individual be subjected to a temperature of sixty degrees Fahrenheit it will be found that the blood vessels of the nasal chambers will possess a certain caliber, the resultant of the cooperative action of the vasodilatator and vasoconstrictor nerve centers. This caliber will permit the passage of a definite volume of blood with a definite velocity in a unit of time, carrying with it a certain volume of heat. By reason of the difference in the temperature between that of the blood (ninety-eight or ninety-nine degrees Fahrenheit) and that of the air passing into and through the nasal chambers—viz., thirty-eight degrees Fahrenheit—heat will at once be radiated into the chambers, and being absorbed by the air will raise its temperature. The degree

*The ganglia—the sphenopalatine, the otic and the submaxillary—though anatomically associated with the trigeminal nerve, are not integral portions of it; they belong to the autonomic system of nerves and are associated with and constitute a part of the vasomotor and secretory systems of nerves. They possess neither sensor nor motor functions.

to which the temperature will be raised is difficult to determine, as the absorption of heat will depend very largely on the rapidity and depth of the inspiratory movement, and hence the duration of the stay of the air in the chambers.

If the external temperature should fall to thirty degrees Fahrenheit, the arterioles of the nasal mucous membrane, as well as those of the general cutaneous surface, will contract as a result of the now preponderant action of the vasoconstrictor center, due to the stimulation of the afferent nerve endings by the cold air. This would of course diminish the thickness of the blood stream, a condition which at first glance would diminish heat radiation. But it must not be forgotten that though the thickness of the blood stream is diminished, its speed is increased, so that in a unit of time an equal, or perhaps even a greater, volume may pour through the vessels, carrying with it the same, or perhaps greater, volume of heat. Owing to the now greater difference between the temperature of the blood and that of the inspired air—viz., sixty-eight degrees Fahrenheit—the speed with which the heat is radiated will be increased, and, therefore, the temperature of the air will be corresponding raised. When the temperature of the air returns to sixty degrees Fahrenheit, the former condition of the blood vessels will be reestablished.

Should the external temperature be raised to ninety degrees Fahrenheit, the blood vessels will dilate, the thickness of the blood stream will be increased and its speed diminished. This condition is favorable to heat radiation, but owing to the slight difference between the temperature of the blood and that of the air, the speed with which heat is radiated is correspondingly decreased.

With regard to the secretory mechanism, similar statements may be made. It is well known that a diminishing volume of blood flowing into the capillary vessels surrounding glands decreases the secretion, while an increase in the volume of blood increases the secretion—other things being equal. These two phenomena are always associated in the production of secretions by glands in general. Air that is warm ought, therefore, to increase the vascularity and also the secretion. Under these conditions, however, the additional amount of moisture necessary to approximately saturate the air may render the increase in secretion unobservable. In warm and moist

air the secretion increase may be noticeable, because the air, having a high water content, does not take up the water in the secretions, and the excess is drained out of the nostrils.

Air of a low temperature, on the contrary, should theoretically diminish the secretion, by reason of the diminished vascularity and the local action of the cold air on the mucous membrane. Nevertheless, it is within the range of observation and experience that the secretion on a cold day is not infrequently very much increased. As to the manner in which this is accomplished there is much obscurity. A possible explanation for the fact may be found in the effects that follow the action of currents of cold air passing over more or less suddenly exposed regions of the body. It is a familiar observation with many people that the exposure of the cutaneous surface of the head and ankles by the removal of the customary coverings is followed promptly by an increase in the nasal secretion, and possibly a local increase in vascularity. The increase in secretion trickling over the surface of the mucous membrane stimulates the endings of the trigeminal nerve, thus giving rise to the sensation and the reflex act of sneezing. A similar condition is established by the plucking of hairs from the inside of the nostril, or by the inhalation of snuff. It is a possibility, therefore, that though the inhalation of cold air tends to diminish secretion, it at the same time tends to increase secretion reflexly through its action on cutaneous nerves. The effects produced might be regarded as the resultant of the action of two antagonistic forces.

Should the vascular and secretory changes continue, it is possible that under changed conditions of nutrition and lowered vitality the mucous membrane might become the seat of bacterial invasion and activity, with the development of the nasal phenomena characteristic of acute rhinitis. This is especially true in those whose nerve centers have been rendered unstable by dietetic or alcoholic excesses, or by the increased production or defective elimination of metabolic products.

DIRECT OR CEREBRAL EXCITATION OF THE CENTRAL MECHANISM.

That psychic states of an emotional character can influence the secreto- and vasomotor centers in the lower regions of the nerve system, has been observed by everyone from personal

experiences and abundantly demonstrated by experiment on mammals by Pavlov, Cannon and others. It is only necessary to recall the effects of the psychic states on the secreto- and vasomotor centers regulating the flow of saliva and gastric juice; in similar centers regulating the secretions and vascular phenomena of the sexual organs. Evidence has been recorded that psychic states of a sexual character have and do cause similar phenomena in the nasal mucous membrane—viz., increased vascularity, increased secretion and erection of its tissues. It has been reported by J. N. McKenzie that a patient of his always had an attack of acute rhinitis after a period of sexual excitement.

These facts, taken collectively, show that the physiologic conditions of the nasal chambers require for their maintenance a complex nerve mechanism; a mechanism which, however, may be easily impaired and rendered unstable by internal causes, whereupon it acts in an abnormal manner, and thus lays the foundation for the development of pathologic states of longer or shorter duration.

XXXIII.

ON THE RELATION OF DISEASES OF THE ACCESSORY SINUSES TO DISEASES OF THE EYE, ESPECIALLY IN CHILDREN, WITH A REPORT OF TWO CASES.

Report of a Case of Orbital Abscess and One of Thrombosis of the Cavernous Sinuses.

By J. H. Bryan, M. D.,

Washington, D. C.

The relation of diseases of the accessory sinuses to diseases of the eye is now well established. In the past I have presented several communications on this subject, and reported cases showing the influence of the various affections of the accessory sinuses on the orbit and its contents. Most if not all of these cases occurred in adults. But diseases of the sinuses occurring in children have been only slightly considered, for the reason that these cavities in the very young are supposed to be so small they could not be the seat of an inflammation sufficiently severe to cause any serious disturbance with the eye.

These cavities vary in size in children as in adults, and in many subjects are quite well developed; and when severely inflamed that they have a serious influence on the eye is a well established fact, as will be demonstrated in the report of the following interesting cases:

Case 1. Suppurating Ethmoiditis Causing an Orbital Abscess in a Child Eighteen Months Old.—W. M., aged eighteen months, was admitted to the Episcopal Eye, Ear and Throat Hospital in March, 1914, on the service of Dr. John Burke, with a very marked exophthalmos on the left side.

His mother stated that during the recent epidemic of influenza all the members of her family had been more or less seriously affected by the disease. About a week after this child had been infected, his left eye commenced to protrude from the orbit, and there was a severe swelling of both lids, especially the upper. The family physician applied lead and

opium wash to the parts, but no relief being obtained, he brought the child to the hospital.

On admission his temperature was 104°. There was some secretion flowing from the left nostril, and there was a very marked bulging of the left eye downward and outward. The lids and conjunctiva were edematous, and the periauricular glands were enlarged. As the child could not be controlled, it was not possible to make a satisfactory intraocular examination.

The following day I was asked by Dr. Burke to see the case, and we decided it was a case of orbital abscess resulting from an infection through the ethmoid cells. The radical operation was then done, the incision commencing at the junction of the middle and outer third of the supraorbital ridge, and was carried inward and downward along the inner border of the nose below its middle. The periosteum along the inner wall and the corresponding parts of the roof of the orbit was stripped from the bone, and in doing so a large quantity of pus was evacuated. The whole of the inner wall of the orbit was removed back as far as the sphenoid. The ethmoid cells were found to be badly diseased, especially the middle and posterior portion, and from the condition I found, it was apparent the orbital abscess resulted from a direct infection from the middle and posterior ethmoid cells. The amount of pus evacuated was enormous, when we take into consideration the age of the child and the stage of development of these parts at this age. The abscess having been thoroughly evacuated, a strip of iodoform gauze was placed in the orbit back of the eye and brought out through the nose, and a small gauze drain was placed just inside of the inner canthus, and the external wound closed by interrupted sutures.

This child made an uninterrupted and quick recovery, the eye, gradually receding, soon assumed its normal position. This is the first case of such a marked abscess of the orbit resulting from sinus disease in one so young that I have met with, and in the light of my past experience with these conditions, I believe it is most unusual.

Case 2. Chronic Abscess of the Posterior Ethmoid Cells and Sphenoidal Sinus Causing a Marked Exophthalmos in a Child Eleven Years of Age.—Robert C., aged eleven years, negro, was referred to me from Dr. Henning's clinic on the eye serv-

ice of the Episcopal Eye, Ear and Throat Hospital, November 2, 1915. Giving a history of the present condition having existed about six weeks, on examination the left eye was found bulging to a marked degree downward and outward. The bulging was so great that the lids did not completely cover the ball. There was no secretion from the nose or nasopharynx. Rhinoscopic examination showed only a moderate swelling of the middle turbinal on the left side. Probing in the anterior and middle ethmoid region failed to reveal any caries or necrosis of the bone, and no secretion followed irrigation of the nose.

The following are the findings after an X-ray examination. Stereo plates in the lateral and posterior anterior positions showed the frontals to be normal; the ethmoids clear on both sides; the maxillary sinuses clear on both sides. The left orbital cavity is apparently filled with a dense mass which seems to be confined to the orbit.

In the light of the X-ray findings, and in the absence of any catarrhal condition present, and the negative history of any previous catarrhal state, it was thought we had to do with a growth in the orbit back of the eye. I therefore decided to explore the orbit. An incision along the orbital ridge was made, commencing at its outer third and extending down on the inner side of the nose to its lower third; the orbit was exposed and the periosteum covering its inner wall was elevated. At the junction of the middle and posterior portion there was a decided bulging of the ethmoid toward the orbit. With a probe the cells were perforated, and a large quantity of pus was evacuated. The whole of the inner wall of the orbit, including all the ethmoid cells, were removed as far back as the sphenoid, and in doing so I found a large abscess involving the posterior ethmoid cells and the sphenoidal sinus. The sphenoidal cavity was unusually developed and filled with thick, creamy pus. All diseased bone and purulent secretion having been thoroughly removed, an iodoform gauze packing was placed in the sphenoid and ethmoid regions, one end being brought out through the nose and the external wound closed by interrupted sutures. At the end of the second day the gauze drain was removed and the nose gently irrigated with a saturated solution of boracic acid.

The patient made a quick recovery, the eye gradually reced-

ing within the orbit, and at the end of the ninth day he was discharged from the hospital. About ten days later I saw the patient again in the eye clinic, and his eye had assumed its normal position and the vision was perfectly normal.

Photograph No. 1 illustrated fairly well the amount of exophthalmos when I first saw the patient. This case is interesting because of the deceptive character of the X-ray findings, the absence of nasal symptoms, and the unusual size of the sphenoidal sinus in a subject so young.

Case 3. Thrombosis of the Cavernous Sinuses.—This patient, a man of thirty-six years of age, was first seen by Dr. Henning about six o'clock in the evening of November 3, 1915.

The history Dr. Henning received from the family physician was that the patient in June had an infected hand which was slow in healing, and this was followed by a series of furuncles in both external auditory canals, all of which finally healed under appropriate treatment. During the latter part of October a pustule appeared on the nose, and was opened by the patient. The following day he was seized with severe pain over the left eye, followed soon afterward by exophthalmos on the same side.

When Dr. Henning first saw the patient there was a marked bulging of the left eye, with commencing conjunctival edema and diplopia. The optic nerve head showed a blurring of its edges, and the veins of the retina were a trifle larger than those of the right eye. At this time the right eye was normal in every respect. The temperature was 104°. Examination of the nose showed a moderately swollen middle turbinate on the left side. Irrigation of the nose brought away some thick blood stained mucopus. As the patient was in a very serious condition and growing rapidly worse, he was removed to the Episcopal Eye, Ear and Throat Hospital. At nine o'clock the following morning the exophthalmos was more marked in the left eye, the conjunctiva was so edematous that it overlapped the cornea and protruded between the lids. There was also a commencing exophthalmos on the right side. The temperature was 104.6°. Although quite dull, the patient was able to comprehend the questions put to him, and stated that he was free from pain. At noon I saw the patient in consultation

with Dr. Henning, and found the exophthalmos on both sides very marked, conjunctiva protruding between the lids and exuding a bloody secretion. Examination of the nose showed the turbinals on both sides swollen and intensely congested. I probed into both sphenoidal sinuses, and on irrigation brought away a large quantity of thick yellow mucopus.

Later in the afternoon, under ether anesthesia, I removed the middle turbinals and opened both sphenoidal sinuses, finding some mucopurulent secretion in both cavities. Examination of the secretion taken from the sphenoidal sinuses showed both streptococci and staphylococci present. A lumbar puncture showed the spinal fluid under great pressure, filled with pus cells, roughly estimated at forty thousand per millimeter. Polymorphonuclear leucocytes predominated. The condition of the patient grew rapidly worse, and he died at 10 p. m.

Postmortem examination, the head only examined, showed all the sinuses contained dark and semifluid blood; small hemorrhages on the right half of the cribriform plate of the ethmoid bone. Vessels of the pia mater generally engorged, the basillar surface of the brain showed fibrin and a purulent exudate under the pia, especially on the insula and adjacent opercula. A microscopic examination showed the vessels of the brain engorged. Beneath the membrane was a thin layer of brown material, intimately adherent to the brain substance beneath. On section the brain surface showed brownish points near the surface; the brown exudate under the membrane showed fibrin, red blood cells and mono- and polymorphonuclear round cells.

Photograph No. 2 shows a typical picture of the conditions found in thrombosis of the cavernous sinuses. This case is not only rare but interesting, and was characterized by a sudden onset in which the initial symptoms were ophthalmic and then meningeal, and death within forty-eight hours. When we take into consideration the free anastomosis of the veins of the nose, pharynx, mouth and face, with the veins of the orbit and the cavernous sinuses, and the absence of valves in the veins, we can appreciate the readiness with which the infection was carried from an insignificant pustule on the face to the cavernous sinuses and then to all the sinuses of the brain.

Photograph No. 1.

Photograph No. 2.

XXXIV.

TWO CASES OF CHRONIC PANSINUITIS ASSOCIATED WITH SYSTEMIC INFECTION.

By George E. Shambaugh, M. D.,

Chicago.

I venture to record the two following cases of pansinuitis, first, because both serve to illustrate the difficulties occasionally encountered in attempting to eradicate the infected foci, and second, because both cases were associated with a serious systemic infection, which presumably took its origin from the infected sinuses.

Case 1.—Miss W., aged thirty-five years, consulted in first in September, 1910, complaining of purulent discharge from both sides of the nose and into postnasal space. She was suffering also from disturbed digestion, which she attributed to the swallowing of the secretion from the nose, especially during sleep. She gave the following history of her nasal symptoms:

The trouble began from an infected upper molar, from which her dentist, in 1895, removed an old filling and killed the nerve because of a severe toothache. In 1897 this tooth was crowned. After this the tooth remained sensitive, and she detected a bad odor from the pus, which continued to escape about the crown.

In 1900 she began to suffer from pain over the maxillary sinus, and noticed for the first time purulent discharge from the nose. In March, 1903, had the crown removed and the diseased root extracted. In April of same year, because of continued pain in upper jaw, was given chloroform and a fragment of necrosed bone removed from the upper jaw. The following June, under chloroform, an opening was made into the antrum from the canine fossa and extended through into the nose. Again in October, same year, a similar operation was undertaken, under chloroform, because of the continuance of the purulent discharge.

After these operations she continued to have a purulent discharge from the nose, and suffered from symptoms of persistent head cold and more or less severe headache, but had nothing more attempted in the way of treatment until 1909, when for the first time she came under the care of a rhinologist. The previous work had been done by a general surgeon. She was again given a general anesthesia, when the antrum was opened from the canine fossa and a satisfactory permanent opening made into the inferior meatus of the nose. Later, under local anesthesia, the middle turbinated bodies, both sides, were removed and the ethmoid cells drained. The patient received a great deal of relief from these later operations, especially from the headache and pain in the region of the frontal sinuses.

No further operation was attempted until she consulted me in September, 1910. An examination at that time discovered creamy pus in both nasal chambers. On the left side pus came from the sphenoid sinus as well as from the frontal sinus and some anterior ethmoid cells. On the right side pus was washed from the antrum and could be traced as coming from the frontal sinus as well as the anterior ethmoid cells. It was also possible to trace pus coming from the right sphenoid sinus. At this time an operation was undertaken, under local anesthesia, on the left side of the nose. The anterior wall of the sphenoid sinus was removed. The remaining anterior ethmoid cells, from which pus was exuding, were removed, and the opening into the frontal sinus was enlarged by means of a frontal sinus rasp.

In February, 1911, patient returned to the city. Discharge from the left side of the nose had become much less, but could still be traced to the frontal sinus and to a narrow canal extending directly backward from a point just below and to the outer side of the floor of the sphenoid sinus. Into this canal a probe could be passed a full centimeter. This infected fistula seemed to be nothing else than the Vidian canal.

She consulted me again in May, 1911. A month previous she had suffered from an attack of articular rheumatism, which began in the ankles and involved later both knees, the hips and elbows. The joints were distinctly swollen. Under local anesthesia an operation on the right side of the nose was under-

taken. A large opening was made from the middle meatus into the antrum. A large posterior ethmoid, full of thick pus, was opened. The anterior wall of the suppurating sphenoid sinus was removed.

In October, 1911, patient was seen again. The rheumatism had entirely disappeared. She had been much relieved by the last operation. The headache, which had annoyed her, had entirely disappeared. The discharge from the nose had been greatly reduced. On the right side of the nose the only place from which pus was still exuding was the frontal sinus and from the fistula above. On the left side pus was still coming from the frontal sinus and from the fistula described above. On irrigating the right frontal sinus the water passed through to the left side of the nose, showing a perforation in the frontal septum. The opening into the right frontal sinus was enlarged at this time by means of the frontal sinus rasp.

The condition when patient was last seen in March, 1915, was as follows: A small amount of pus coming from both frontal sinuses and from the fistula on the left side. In spite of the removal of a large part of the inferior turbinated bodies, as well as the whole of the middle turbinates, and the exenteration of the ethmoid labyrinth, both sides, there has been no development of atrophic changes or crust formation other than that forming at the points at which pus was appearing.

Case 2.—Miss M., aged twenty-five years, came under my care in February, 1914, suffering from severe multiple arthritis. She gave the following history: Since childhood she had suffered more or less from nasal trouble. The symptoms were extreme liability to head colds, annoying discharge from the nose, with subsequent irritation of the throat and attacks of acute laryngitis. From 1897 to 1899 she had a number of operations on the sinuses, one of which consisted in the opening of the left maxillary sinus through the socket of a molar tooth. In this opening she wore a tube for about a year, when she removed the tube and allowed the opening to granulate shut. At the time when the antrum was drained an intranasal operation was done to relieve a frontal sinus abscess which was causing excruciating pain in the region of the left eye. For two or three years following this operation had a probe passed from time to time into the frontal sinus.

No further operating was done on the nose until in Sep-

tember, 1912. During this time she continued to be annoyed by the nasal symptoms recorded above. In August, 1910, she had suffered from a severe pain in the left maxillary sinus, which lasted several days only. On January 18th, of the same year, she was seized with a violent attack of acute articular rheumatism. The arthritis still persists and involves every joint in her body.

In October, 1912, work was begun on the nasal sinuses, in the hope of removing the possible focus for the serious systemic infection. The ethmoid cells, left side, were broken down and adhesions were found between the remnants of the middle turbinated body and the septum, the result of some previous operation.

In December, 1913 the left maxillary sinus was opened through the middle meatus and pus washed out. Irrigation of the right antrum at this time disclosed no evidence of pus infection.

I saw the patient first in February, 1914. She was suffering greatly from the arthritis, which involved every joint in the body. She was unable to walk, every joint was swollen and painful. She experienced at times acute exacerbation of the infection, now in one joint, now in another.

The examination of the nose disclosed completely atrophied inferior turbinated bodies. The middle turbinated body, both sides, was absent. There were some crust formations here and there in the region of the ethmoid, more marked on the right side, but no evidence of ozena. A large opening was found into the antrum on the left side. No free pus was found in antrum. The anteroposterior radiogram gave no evidence of a frontal sinus either side. It was with difficulty that a small probe could be passed into the opening of a small frontal sinus on the left side. The withdrawal of the probe was followed by a drop of pus. From the right maxillary sinus a quantity of thick mucopurulent secretion was washed out. A large opening was made at this time from the middle meatus into the right antrum. The patient has since been able to irrigate both maxillary sinuses herself, and both have remained practically free from secretion, except for a short period following acute head colds.

The left frontal sinus has been repeatedly irrigated and, as a rule, a small amount of granular pus washed out.

In January, 1915, by exploring carefully with a probe the locality in the right nares where crusts were seen to form, a large posterior ethmoid cell, filled with thick creamy pus, was discovered and drained. At the same time the opening of the right sphenoid sinus was discovered in the same way and the anterior wall removed. The sinus was filled with the same kind of pus. Two weeks later, by the same method of exploring with a probe the region in the front of the nose where crusts were forming, an ethmoid cell was discovered, also filled with thick pus. I was not able to discover an opening to a frontal sinus on the right side.

In February, 1915, two small ethmoid cells on the left side were found secreting pus and were drained. The crust formation on both sides of nose much improved. Present condition of patient, as far as the nose is concerned, seems to be quite satisfactory. There is no crusting, in spite of the entire disappearance of the inferior and middle turbinated bodies and the exenteration of the ethmoid. The patient feels more comfortable when she irrigates the nose once each day, but washes out very little secretion and no crusts. A careful examination fails to discover any distinct evidence of pus in any of the sinuses. The general condition of the patient is not at all good. She is free from acute exacerbations of the joint infection and the associated rise in temperature, but the inflammatory changes in the joints are apparently undergoing secondary fibrous changes, which is resulting in greater and greater rigidity. These changes are producing, probably through pressure, more or less continuous pain in all the joints in the body.

Chronic infection in the nasal accessory sinuses is rarely limited to a single sinus. The process not infrequently begins as an infection in a single sinus, as when the antrum is infected from a diseased tooth, but, as a rule, other sinuses become sooner or later involved. This is especially true of those accessory sinuses which open into the middle meatus. The infundibulum often forms a more or less closed trough, which opens at its anterior end into the frontal sinus, and at its posterior end into the maxillary sinus. This trough receives drainage as well from the anterior ethmoid cells. By means of the infundibulum, pus secreted from either the frontal sinus or the

anterior ethmoid cells drains directly into the maxillary sinus when the head is in the upright position. On the other hand, when reclining, pus from the antrum is conducted by the infundibulum directly into the frontal sinus. I have occasionally encountered chronic infection limited to the sphenoid sinus, but it is much more common to find combined empyema of all the accessory sinuses on one side.

The method of choice in dealing with chronic accessory sinus infection, whether the process involves one or several sinuses, is by carefully conducted intranasal operations performed under the use of a local anesthesia. The operations conducted in this way are safer, and are, as a rule, as efficient as under a general anesthesia. The one sinus most difficult to approach by the intranasal route is the frontal, and here the disfiguring scar always left after an external operation prohibits this method of operation except for the relief of an impending intracranial complication and after all intranasal methods have failed to relieve the pressure and pain from the confined pus.

XXXV.

A REPORT OF TWO SEVERE CASES OF ULCERO-MEMBRANOUS ANGINA.*

By Ira Frank, M. D.,

Chicago.

There occurred recently in my service two unusual and severe cases of ulceromembranous angina, which were of interest not only because of their severity, but also because of the widely differing clinical forms which they assumed. The contrast of these two cases, which I was fortunate to be able to study simultaneously, was so marked—considering that they both represented the same disease, with presumably identical etiologic factors—that I feel that a brief report may be of interest to you. Moreover, a rather diligent search through the literature has shown a marked dearth in the number of serious and fatal cases reported, and has further revealed the existence of debatable points in etiology and treatment. It is, therefore, that I present these two cases in the hope of provoking a general discussion and a recital of personal experiences.

Case 1.—L. S., male, aged forty-eight years, a peddler, was admitted to my service on June 4th. He gave a meager history, which indicated that five weeks previously he had suffered with a peritonsillar abscess which when opened had discharged a large quantity of pus. Some time after admission he was able to tell us that following this evacuation the symptoms continued without remission, though there was no further formation of pus, and his condition grew more serious day by day. He presented himself at the hospital in a pitiful condition, unable to swallow or to open his mouth beyond a one-half inch separation of the incisors. He was weak and emaciated, having lost approximately twenty-five pounds since the onset of his illness.

General physical examination negative. Examination of the

*Read before the Chicago Laryngological and Otological Society, December 21, 1915.

mouth revealed an area of deep necrosis on the right side of the soft palate, oval in outline, extending from and involving the tip of the upper pole of the tonsil, anteriorly to the hard palate, and mesially to the midline. The area involved was composed of a greenish gray, foul-smelling slough, which was easily removed with very little bleeding, and exposed an ulcer one to one and one-fourth inches in diameter. The base was not raised and only slightly injected. The edges were irregular and vertical, with a punched out appearance. The floor was incompletely formed; a green seminecrotic slough, with a perforation through the soft palate large enough to permit a fair view of the superior portion of the nasopharynx.

The clinical diagnosis, from the picture presented and the history obtainable on admission, was not easy—lying between carcinoma, tertiary syphilis and Vincent's angina. Direct smears from the depths of the ulcer showed pus, fibrin, pneumococci, a fusiform bacillus and spirillæ, with the latter organisms predominating.

The bacilli were five to ten microns long, with tapering ends and homogeneous body. The spirillæ averaged ten microns in length, stained deeply with anilin gentian violet stain. The spirilla contained an irregular number of spirals. Aerobic cultures negative, pneumococcus and staphylococcus being present. Blood findings were normal. Wassermann reaction, taken twice, was negative.

A piece of tissue from the floor of the ulcer was submitted to the laboratory for microscopic examination, and the following report was returned: "No malignancy evident. A suppurative process with marked necrosing tendency. Pus cells numerous, with a small round celled infiltration. Infective cellulitis with gangrene."

The patient was given a frequent gargle of peroxid, with daily deep injections of sodium cacodylate in gradually increasing quantities. It was necessary to administer opiates a number of times after admission, on account of his severe pain.

On the third day of his stay at the hospital the first signs of improvement were noted. This became gradually more pronounced, interrupted only by symptomatic remissions which were controlled by sedatives. There was no evidence of further spreading of the process, and by the fifth day there ap-

peared definite signs of healing in the ulcer. The edges assumed a more normal appearance, and removal of the reforming necrotic center revealed a rapidly healing base.

On the eleventh day the patient was free from pain and the cavity was markedly diminished in size, with cicatrizing edges. The course of the entire process was ten weeks, six of which were spent in the hospital. The patient was discharged in excellent condition. The soft palate was distorted by a firm scar, the center of which contained a narrow fistula, admitting a small silver probe, leading up into the posterior nares.

Case 2.—E. S., female, aged twenty-two years, was admitted to the hospital on June 21st. For one week previous she had complained of severe sore throat, with almost total dysphagia and the usual constitutional symptoms accompanying a severe throat infection. Cultures taken during this period were negative, and smears from the throat showed large numbers of both fusiform bacilli and spirillæ. She was given peroxid gargle without any alleviation of her symptoms, and because of her practically complete inability to take nourishment, she was placed under hospital care.

General examination was negative. On inspection the right tonsil was found covered with a greenish gray membrane which extended beyond the tonsil anteriorly and posteriorly, hanging in small islands on the pharyngeal wall. The membrane was adherent and tough, and rested on an angry red base. It was removed with difficulty. The uvula, soft palate and pharynx were edematous. A large piece of thickened membrane was removed and sent to the laboratory for examination. The report showed necrotic tissue with hyalin degeneration, considerable round celled infiltration and occasional patches of pharyngeal mucosa, surrounded by coagulated débris. Laryngeal examination revealed marked redness of the larynx, especially of the epiglottis, and no edema. Nasal examination negative. Schick reaction negative. Urinalysis negative. White blood cells, 24,000. Differential: Seventy-six per cent polynuclears, two eosinophiles, eighteen mononuclears. Temperature mean, 102°. A gargle of peroxid was ordered for frequent use, and also twice daily deep injections of sodium cacodylate in three-fourths grain doses.

There was no appreciable symptomatic improvement fol-

lowing the introduction of this treatment, though examination showed a less acute process on both tonsil and pharynx, and the membrane covered smaller areas than when first seen. The patient complained more bitterly of pain. She became hoarse, was unable to talk above a whisper, and no manner of coaxing or scolding would induce her to swallow more than a few drops of milk or ice water at long intervals. Deglutition caused severe attacks of coughing which were pitiful to witness. She complained of an intense substernal burning pain, lasting fifteen to twenty minutes after swallowing minute quantities of water. An attempted laryngeal examination on the second day showed the presence of patches of membrane and edema of the epiglottis. Examination of the cords was unreliable, because of strenuous objection by the patient.

The situation becoming more and more serious, an effort was made to stay the process by increasing the cacodylate dose to two grains, the intense dysphagia and substernal burning with the increasing signs of laryngeal involvement indicating an extension into the esophagus and larynx.

The urine was at all times scanty, and on the third day showed very positive albumin, hyalin and granular casts. Fluid intake less than eight ounces, practically all of which was water.

Late in the second hospital day the patient exhibited signs of collapse. The cause being attributed to lack of nouishment, and because she was unable to retain nutriment per rectum, five per cent glucose solution was administered intravenously. On the third day the picture remained unchanged, slight improvement noted in the pharyngeal lesions. The urine was scanty and test showed a heavy deposit of albumin. Signs of gradual collapse continued. The patient became irrational and refused to swallow any nourishment. The fourth day was marked by no change in the condition. Toward evening the patient suddenly had a severe retching spell, lasting nearly five minutes, at the end of which she vomited a piece of membrane about four and one-half inches long and one inch wide, thick, with straight smooth sides and irregular ends—evidently a large portion of a cast of the esophagus. Toward morning of the fifth day the patient coughed and suddenly became dyspneic and cyanotic. Emergency tracheotomy was about to

be performed when a piece of membrane was forcibly expelled, relieving the symptoms as suddenly as they had appeared. This specimen was three inches long and about half an inch wide—no doubt, a good portion of the tracheal and laryngeal membrane.

There was no appreciable alleviation of symptoms following these incidents. For four days following she had an average intake of five to seven ounces of fluids, and a total twenty-four hour excretion of eight to ten ounces. The pulse became irregular and slow, and examination showed a toxic myocarditis present. Mental symptoms became prominent; the patient satisfied herself that she was going to die, and it was impossible to obtain her cooperation in any therapeutic measure. She exhibited a taste perversion which was marked. Water caused her intense agony when swallowed, but she was certain that sarsaparilla "pop" would go down easily. We procured some, which she drank with apparently not much difficulty. The idea of impending death was so fixed that she prevailed on her people to take her home to die. Exitus occurred on the second day after leaving the hospital.

These cases, both clinically very malignant, present markedly differing types. The first was ulcerative and necrotic, a deep slowly extending destructive process, with symptoms relating only to movement of the affected parts. There were no systemic effects, not even an elevation in temperature, and the lesion responded readily to treatment. The second case was strikingly different. The process was primarily membranous, with only a slight tendency to superficial ulceration. The membrane spread rapidly—evidence of its migration in both the trachea and esophagus being given above. Symptomatically the case was pronounced toxic in features, fever varying from 99° to 103°, nephritis and myocarditis being complications.

What influence, working on the same etiologic factors, produces this widely differing clinical picture? It has been suggested that the course of the disease is modified by the relative numbers of spirillæ and fusiform bacilli present—that predominance of one type of organism or the other produces the varying clinical picture. Such a condition we were unable to prove in these two cases.

It may be suggested that the slow ulcerative type is a chronic

infection, whereas the membranous type is an acute or fulminating example of the same disease. I am inclined to hold the latter view, accounting for the ulcerative form as a low grade infection, due either to attenuated organisms or an immunizing protective effort of the body. Examination of the microscopic specimens in the first case certainly show such a condition—the lesion was well limited by a leucocytic wall.

The fulminating character of the second case may well be ascribed to infection with virulent organisms in a host physically unfit to offer resistance and combat the process. As to a comparison of the clinical course, little explanation is necessary. The first case produced no more systemic results than would a walled off abscess; the second patient showed systemic effect due to (1) the virulent infection, (2) absorption in the large area involved, and (3) consequent secondary infection.

XXXVI.

EAR AFFECTIONS AND DIABETES.*

By Chas. Zimmermann, M. D.,

Milwaukee.

Although we do not know of any ear disease peculiar to diabetes, there is no doubt that diabetes has an unfavorable influence on the course and healing of ear affections, imparting to them a certain anatomic and clinical character, and, especially with regard to operative measures, its presence demands the most serious consideration. On the other hand, ear diseases may elicit the chief symptom of diabetes, viz., glycosuria. (Grunert,[1] Frey,[2] Furet.[3])

To begin with affections of the external ear, obstinate eczema of the auricle and the external meatus with annoying pruritus has been observed. Moos reports such an experience on himself. This is in accordance with the skin affections on other parts of the body in diabetes. More severe are furuncles of the external meatus, which often heal very slowly (several weeks—Srebrny[4]), even after incision, and, according to Wolf, have a great tendency to relapses or may lead to coma. Kipp reported a case in a child, affected with diabetes, who in spite of extensive incisions died after twelve hours in coma. In a diabetic patient of Eulenstein,[5] a woman, aged fixty-six years, a severe phlegmon developed after a circumscribed external otitis. Large incisions were made to the bone, which was denuded of periosteum, with free drainage, but the patient died seven days later in coma. Grunert[6] found in Schwartze's clinic in furunculosis diabetes not more frequently than after the indiscriminate urine examinations of all stationary ear

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patients, and does not consider diabetes as a predisposing element for the constant recurrences.

Miot[7] states that diabetics suffer at times from ear troubles of varying intensity and duration, which are caused by swelling of the eustachian tube. Some of the cases are curable, in the others the tubes are permanently obstructed and impermeable for air or bougies. (The constant current allays the congestion of the mucous membrane, and renders possible the introduction of bougies and the application of chemical and galvanic cauteries.)

Valentin[8] described that in diabetes oidium albicans may enter the eustachian tube from the nasopharynx to the tympanic cavity and produce otitis media with perforation of the membrana tympani.

Of all ear affections in diabetes, purulent otitis media has been observed most frequently. According to Koerner's statistics,[9] and a critical analysis of forty-six cases by Eulenstein, partly observed by himself, partly from literature,[10] diabetic persons have a greater tendency to suppurations of the ear and temporal bone than nondiabetics, and acute otitis media in diabetics leads much more frequently to diseases of the bone, to extensive caries and necrosis, than in nondiabetics.

Grunert[11] had the same impression from his experiences in Schwartze's clinic. Although the formerly held opinion of a specific diabetic otitis has been refuted as erroneous, it is certain that the greater vulnerability of the tissues of the body in diabetes predisposes to the occurrence of acute otitis media and retards the healing process, so that the affection is apt to become chronic.

This predisposition in diabetes is given by the presence of sugar in the blood and tissues, furnishing a very good culture soil for microbes. It is well known from experimental bacteriology that animals that are ordinarily proof against certain microbes can be successfully inoculated with these after grape sugar has been introduced into the body and an artificial diabetes produced. Thus Bujwid created local gangrene of the skin of animals by injecting very slight, in themselves harmless, quantities of staphylococcus aureus under the skin and simultaneously grape sugar into the auricular vein. Likewise, von Mering and Minkowski[12] observed that their glycemic

dogs, after extirpation of the pancreas, inclined to suppurative processes and with one exception never showed aseptic healing. Also Ernst[13] proved clinically and experimentally that, diabetes creates a disposition and favorable soil for the growth of the most different lower fungi. Hence, one must assume that the cells of the tissues must be changed by the sugar, so that they do not offer sufficient resistance to morbid agents.

This is further due to lack of nutrition from early arteriosclerosis in diabetes, as first pointed out by Israel. Also Partsch[14] found in diabetes endarteritis of the smaller arteries, with diminution of the lumen, calcification and even ossification of the media, which play an important rôle in the etiology of spontaneous diabetic gangrene. According to the histologic investigations of Hildebrandt,[15] diabetes leads early to degeneration of the contractile vascular walls, secondarily to a proliferation of connective tissue. Clinically he proved this by examination with Roentgen rays. His skiagraphs showed intense sclerosis of the arteries and veins. Intestinal hemorrhages, extravasation in skin and mucous membranes—e. g., of the mouth—epistaxis, hemorrhagic otitis media, subcortical hemorrhages, described by a number of authors (e. g., Sendziak[16]), find their explanation in these vascular alterations. Hence Garel and Armand,[17] who reported thirteen cases of epistaxis in diabetes, recommend the examination of the urine in frequent relapses of epistaxis.

The occasional serious character of acute otitis media in diabetes is illustrated by a case of Chorazycki.[18] The patient, aged twenty-three years, felt greatly relieved after paracentesis. The next day his condition changed for the worse; he had a low pulse and died on the second day. A case of Heiman,[19] who came to him in the fourth week of an acute otitis media, had temperature of 39.5° C. and three and one-half per cent sugar in the urine. The suppuration ceased after a week, and the perforation of the membrana tympani closed. Two weeks later he became unconscious and died under the symptoms of meningitis.

The following cases of acute otitis media and acute exacerbations of chronic purulent otitis media in diabetes took a favorable course, not much deviating from the ordinary form:

Case 1.—March 12, 1912, I saw in consultation a feeble

man, aged sixty-five years, who had been affected with diabetes for years, on account of intense pain in his left ear for two days from otitis media. There was a slight discharge, and the meatus was very much swollen A large paracentesis relieved him at once, and he made a good recovery.

Case 2.—On December 31, 1892, I was called to a man, aged fifty years, diabetic for years. He was in bed after an operation for empyema of the pleural cavity, and now complained of pain and discharge in left ear. Large perforation in anterior quadrant, and very offensive secretion. Under treatment the discharge stopped February 17, 1893. March 15, 1899, ear ran again after a severe cold; again on June 6, 1899, which soon healed. The patient died a few years later.

Case 3.—H. B., aged fifty-two years, diabetic, who had since childhood a discharge from his right ear, came on July 28, 1914, on account of pain and recent profuse suppuration of this ear. There was a small perforation in anterior portion of the membrana tympani, the posterior portion was bulged forward and the mastoid process was very tender; temperature, 99°. Apparently the posterior portion of the tympanic cavity was shut off by adhesions. I therefore made a large curved incision in the posterior portion of the membrana tympani and up to the perforation, which at once relieved him. The pain in the mastoid subsided, and after a few weeks the discharge from the ear ceased.

According to Koerner, adults, especially men, suffering from diabetes, do not easily recover from an acute otitis media without severe, very rapidly spreading mastoiditis. It is characterized by extensive central destruction of bone in the mastoid process, before signs, tenderness to the touch or swelling, show on the outer surface. Apparently it commences by hematogenous infection of the bone, extends early to the pneumatic cavities, and discharges its pus on the preformed path through the tympanic cavity, and at first has the aspect of mere suppuration of the middle ear. The mastoid may be converted into a large cavity, filled with granulations and pus, covered by a very thin corticalis, and the operation reveals the inner table of the sigmoid sulcus or the middle cranial fossa destroyed to a large extent. In these cases a valuable diagnostic

means of recognizing the serious condition is percussion of the bone, originally devised by Lücke and further developed by Koerner. From the experience of Lucae's clinic, Grossmann[20] reports that out of the cases of acute otitis media which came to operation 72.7 per cent occurred in diabetic, 56.3 per cent in nondiabetic, patients, due to the alteration of the tissues by diabetes, as set forth above.

All microorganisms that cause otitis media were found in diabetic mastoiditis, viz., streptococcus, staphylococcus albus, and diplococcus, which seems to have a greater tendency to spread beyond its original field of infection, and mixed infections. As further complications have been observed: Necrosis with formation of sequestra (Massier,[21] Koenigsbauer[22]), propagation of the suppuration to the dura, thrombosis of the sinus (Leidler[23]), brain abscess (Heimann[24]), periarticular abscess of the maxillary joint (Ephraim[25]), paralyses of the abducens (Luc[26]) and the facial nerves (Castex[27]).

Affections of the inner ear in diabetes have been considered rare. In 1892, Moos[28] wrote: "Little is known about diabetic affections of the labyrinth." Sendziak[29] considers affections of the inner ear in diabetes much rarer than those of the middle ear. They occur in form of vertigo, tinnitus, and progressive hardness of hearing. Heiman, who is of the same opinion, observed a woman, aged seventy-two years, who became deaf in both ears from labyrinthine disease and diabetes. The hearing returned after two weeks' use of Carlsbader water.

Very interesting is the following case of Lermoyez,[30] diagnosed as anemia of the labyrinth: A man, aged forty-three years, diabetic, who had syphilis and formerly had been addicted to alcohol, suffered, after a cure of abstinence, from tinnitus, vertigo, and hardness of hearing for three and one-half years. The reddened promontorium shone through the retracted membrana tympani. Under strict diet the condition deteriorated. Pilocarpin injections were without result. After meals the patient felt heavy in the forehead, but could hear better, and the noises were diminished for from four to five hours. From this Lermoyez inferred an anemia of the labyrinth, which was favorably influenced by the congestion of the

brain occurring during digestion in dyspeptics. The improvement of hearing by inhalation of amyl nitrite supported this supposition.

In some cases the labyrinthine symptoms probably may be due to neuritis of the auditory nerve, an assumption which is admissible if we remember the not rare occurrence of neuritis and atrophy of other nerves in diabetes—e. g., the optic nerve, the sciatic, anesthesiæ and paresthesiæ and neuralgiæ—e. g., of the mastoid (Grunert).[31] This has been corroborated by the anatomic investigations of Steinbrügge, who found in a diabetic individual a hemorrhagic otitis media with microscopically visible extravasations of blood in the labyrinth, at the periosteum of the internal meatus, between the fibers of the auditory nerve and its branches, in the spiral ligament, the endosteum of the vestibulum, and on the neuroepithelium of the ampullæ. According to the conclusions of Friedrich, based on the large material of the ear clinics of Halle and Kiel, there is about one case of suppuration of the labyrinth to every one hundred cases of suppuration of the middle ear. Bezold,[32] however, regards of chronic general diseases diabetes, aside of tuberculosis and lues, as the chief etiologic factor in the proneness of suppuration of the middle ear to advance to the spaces of the labyrinth. This is not surprising, if we consider the tendency of otitis media in diabetes to caries and necrosis mentioned above. The following case may illustrate this:

Case 4.—A sickly looking man, aged thirty years, came to me on April 1, 1898, on account of his hearing rapidly failing within the last three weeks. Right ear was totally deaf. Left ear almost deaf, showed chronic purulent otitis media with very offensive discharge. A broad polypus at the medial wall was removed with curette, and the root cauterized with nitrate of silver. The bone was carious in a large extent. The next day his deafness had increased, and there was some dizziness, but no pain or headache. The parents had noticed the deafness in his third year. Ten years ago he acquired syphilis with secondary symptoms on skin, mouth, tongue and throat.

On April 6th the patient stated that he had diabetes, and showed me a certificate of an examination of the urine made on January 15th by a chemist in New York, who found seven

per cent sugar. Now the amount was the same. The right eye was blind, due to traumatic cataract from an injury twenty years ago. Vision in left eye, 15/70; myopic astigmatism; with his glasses, 15/50. The sight of this eye was failing for about a year. The condition remained the same the following few days. On April 7th he complained of pain in mastoid, without visible signs. The patient then went to New York, and, as I heard later, died on April 20th, but I could not find out under what symptoms.

The occurrence of labyrinthine symptoms in this case after the operation, and in another one after the removal of a large polypus of the inner wall of the tympanic cavity with the snare, without injuring the bone, seems to me due to the fact that the fistula in the carious bone was stopped by the polypus, so that lymph could not, or only very scantily, escape. After removal of the polypus the stoppage apparently ceased and the labyrinthine symptoms set in.

I found a similar explanation in a case of acute purulent otitis media in a diabetic reported by Ephraim,[33] in whom the mastoid operation disclosed a fine fistula in the medial wall of the antrum, leading to a fistula in the horizontal semicircular canal, from which lymph oozed slowly. There were no labyrinthine symptoms. Ephraim attributes this, as also Lucae pointed out, to the slight and very gradual efflux of lymph fluid, as it is very frequent in carious, in comparison to traumatic, defects.

With regard to the prognosis of the operation of mastoiditis in diabetic persons, a perusal of the otologic literature shows discrepant views. While some, mostly older authors, consider it contraindicated, others advise not to operate early, the majority, especially recent authors, regard the operation as a vital indication. E. g., in 1895 Buck stated, before the American Otological Society, that the ten cases of Kuhn and Koerner were all the published instances of mastoid disease occurring in diabetic persons which he had been able to discover. He reported four of his own, of which two died after the operation. He considers it imperative that mastoid operations upon diabetic persons shall be performed at a comparatively early stage of the disease in the temporal bone, that is, before the lateral sinus or the dura mater has become seriously involved.

Since then the subject has been repeatedly discussed, e. g., before the Otologic Section of the New York Academy of Medicine, the Otological Society of New York, the Otological Society of Paris, etc. To mention only a few: Richards[34] operated on nine cases, of which six died of coma; McKernon,[35] thirteen, all in chloroform narcosis; four died. Dench, three; one died. Koerner[36] discussed the operative results on thirty-three cases, discriminating between mild, medium, and severe, of diabetes. None of the light form died; the wounds healed more slowly than in nondiabetic patients, but none developed the severe type after the operation. Also all of the medium kind were cured. Out of the nine individuals suffering from the severe form of diabetes, three died from coma; one from general weakness. All were operated upon in chloroform narcosis. In the mild forms Koerner considers the prognosis good, and the operation also permissible in the severe forms, if there is a vital indication. If, however, the urine shows ferric chlorid reaction, the surgeon must not conceal the possibility that the necessary operation may bring on the threatening coma.

For illustration, I may here report the following case:

Case 5.—Mrs. F. D., aged forty-eight years, came to me in January, 1909, with the history that her left ear had been discharging for about three months. For the last few nights she could not sleep on account of severe pain in ear and head. The left side of the head, temple and mastoid region were intensely swollen, and the auricle was standing off. The walls of the left external meatus were very much swollen, making the otoscopic examination difficult; pus was oozing out, and in the depth there was lively pulsation. As the patient suffered from intense pain, she was at once sent to the hospital, where an hour later I opened the mastoid. After the incision pus oozed. The external surface of the mastoid was discolored to a large extent. After its removal an immense cavity was laid open, filled with granulations. At 4 p. m., temperature, 99°; pulse, 130. The next morning, temperature, 98°; pulse still 130. The patient felt greatly relieved and no pain whatever, but from her laborious breathing I suspected imminent diabetic coma, and asked for the report of the urinary examination. This had been omitted in the rush of getting her ready for immedi-

ate operation. The test now revealed abundant sugar. Upon inquiry, her physician, who had assisted at the operation, but had not mentioned anything of diabetes, told me now that she had it for three years. Bicarbonate of soda, 2.00, every hour, milk and oatmeal, and injections of salt solution were ordered. On the second morning the wound was dressed and found in excellent condition. The patient was conscious and felt comfortable, but the breathing was more laborious. At 6 p. m. the coma was fully developed, the patient was unconscious, and died at 8 p. m.

If I had known beforehand that the patient had diabetes, I would have advised the operation, which was absolutely necessary, but would have guarded the prognosis and mentioned to the relatives the possibility of coma.

If we consult the modern opinions of internists and surgeons as to surgical procedures in diabetes, I cannot do better than to quote from the views, based on unusually large experience, of Kraus, Naunyn, von Noorden, Minkowski, and the surgeons Payr and Karewski, propounded in the common meeting of the Societies of Surgery and Internal Medicine at Berlin.[37] Every patient submitting to surgical treatment must be carefully examined for diabetes. The unfavorable influence of surgical interference may be caused by the psychic shock and the effects of the traumatism on the nervous system, the deleterious influence of the narcotics on the organs participating in the metabolism of the carbohydrates and the acidosis bodies, loss of blood, disturbances of taking nourishment, respiration and circulation, etc. The consequences may be increase of the intensity of diabetes, as shown by the increase of glycosuria observed after mastoid operations, acidosis, acceleration of general weakness and heart failure. The diabetic patient in need of surgical aid is threatened by two dangers: augmented violence of all kinds of septic processes, and the greater predisposition to coma. The first danger can be met by strict asepsis, under which wound healing takes place in diabetes of any form without disturbances, under avoidance of all toxic antiseptics, by adopting the most simple and rapid method of operation, guarding against injuring the tissues by infiltration, anesthesia, caustics, etc. Infectious processes, however, take a malignant course and increase the dangers of metabolic disease.

Every form of diabetes requires careful consideration, according to the strictest principles of internal medicine, before, during, and after operation. These consist in previous dietetic treatment, aiming at reducing the hyperglycemia, prevention of acidosis, preservation of the strength of the patient, and in proper cases abundant ingestion of alkalies for neutralization of the abnormal acids. Here the starvation treatment, devised by Frederick Allen, must be considered, which is simple, safe, and very efficacious in rendering and keeping a patient sugar-free in a much shorter time than it was possible by the old methods.

Coma is the most dreaded enemy of our therapeutic efforts. Neither the light form of diabetes, nor its successful preparatory treatment, insure against coma after operations, which under all circumstances have the significance of a damaging factor, be they ever so slight, as I have observed in the following case:

Case 6.—G. S., a stout, healthy looking girl, aged nineteen years, came to me on August 17, 1903, being blind in both eyes. The strong aceton smell of her breath at once suggested the diagnosis of diabetes, which was verified by the large amount of sugar in her urine. She weighed 117½ pounds, suffered from thirst and voracious appetite. Both eyes showed total diabetic cataract. To prepare her for operation, antidiabetic diet was ordered. When she returned on September 7th, the quantity of urine, which had a strong smell of aceton, in twenty-four hours was still ten quarts. On September 8th a discission of the anterior capsule of right lens was made in cocain anesthesia. The eye bore this very well, but on September 12th, a. m., she had temperature of 101.6°, dyspnea, pulse 96, and headache, foreboding diabetic coma. Afternoon temperature, 102°, pulse 120. The ferric chlorid test revealed diacetic acid in the urine. Large doses of bicarbonate of soda were given every hour. September 13th, the patient was still conscious, but collapsed and cyanotic, pulse more rapid, less tension. The parents, who were sent for, took her home, where she died on September 14th.

Hirschberg had a similar experience.

Postoperative coma may set in after any kind of narcosis and anesthesia, but especially after chloroform. Chloroform

is apt to produce greater acetonuria than ether. Hence Kausch[38] principally denounces chloroform, and considers ether as the narcotic for diabetics. The inhalation narcosis ought to be instituted in the early morning, in order not to prolong unnecessarily the time of the longest physiologic abstinence from food.

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XXXVII.

ESOPHAGOTRACHEAL FISTULA.

By Robert Levy, M. D.,

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Fistulous communication between the esophagus and air passages was recognized many years ago. The literature abounds in numerous cases, the majority of which deal with the congenital variety, to which the greatest attention is paid.

In 1880 Morell Mackenzie[1] collected all the reports of malformations of the esophagus to date, and found thirty-seven examples in which there was a communication between the esophagus and the trachea or one of the bronchi. The similarity of his observations with those of more recent times stamps this condition as one concerning which very little doubt as to diagnosis or prognosis obtains. The symptoms were those that were to be expected when a communication between the food passage and the air passage exists—namely, inability to swallow, violent coughing and choking, the ejection of swallowed liquids, rapid loss of strength and early death from inanition or pneumonia. Other cases of congenital malformation with fistula have been reported from time to time, the total number collated by Zeit[2] in 1912, making fifty-five. A most interesting study of the etiology of these congenital defects may be found in Virchow's Archiv.[3]

Recently, Losee,[4] Finkelstone and Ellis,[5] Schmidgall,[6] and Robertson[7] have added several cases. But while a study of these congenital cases is of great interest and of more or less importance in a consideration of acquired lesions of this description, it is to the latter that I wish particularly to call your attention.

Mosher[8] says that gangrene of the lung due to a fistula between the trachea and esophagus is not an uncommon termination of cases of cancer of the esophagus. He reports the history of a patient who had difficulty in swallowing for six

months, but who suddenly began to cough badly and to have great increase in the difficulty in swallowing even water. He promptly died at the end of a week, and upon autopsy a large fistulous opening was found between the esophagus and trachea. This case was a unique one in Mosher's experience, and must be considered as rather unusual, in spite of the frequency with which cancer involves the esophagus and upper extremity of the trachea and larynx.

The interest attached to a study of these cases, whatever the underlying cause, is in the aid rendered by modern methods in confirming the diagnosis and in the hope that by one of these methods the lesion, when due to other than an incurable affection, may be remedied by some surgical procedure. Unfortunately, the majority of cases of fistula between the esophagus and trachea have been due to carcinoma or tuberculosis. A few cases, however, of less fatal diseases, including syphilis, and some in which the underlying cause could not be determined, have been recorded. In all of the reported cases the similarity in the symptomatology and prognosis to that found in the congenital variety was such as was to be expected. Nevertheless, it is of interest to review the essential features of some of them.

Heddaeus[9] reports the case of a male, aged fifty-eight years, in whom there were found tuberculosis and enlarged bronchial glands. These had undergone suppuration, resulting in a necrotic opening in the anterior wall of the esophagus and posterior wall of the trachea, about the size of the end of one's little finger. The pathologic findings were sufficiently doubtful to raise the question of carcinoma, in addition to the above.

The cases of Riviere[10] were those of children in whom enlarged and caseous glands were associated with tuberculosis of the lungs and intestines. The glands had become adherent to both esophagus and trachea, and the perforation occurring from the trachea to the esophagus permitted the gradual ingestion of tuberculous secretion by the stomach and intestines, thus developing, in addition to the glandular tuberculosis, tuberculosis of the intestines.

Pachina,[11] on the other hand, believes that fistulas are due mostly to tuberculosis of the esophagus, the penetration being from that tube to the air passages. He reports a case in which

four to five centimeters above the cardia, in the left wall of the esophagus, there were two openings leading into lung cavities.

An exceedingly interesting report is that of Dorner,[12] in which an opening was found between the left bronchus and the esophagus, due to necrosis from the pressure of an aortic aneurism. The aneurism in this instance did not burst, as has been the case in others, but pressure necrosis of both esophagus and bronchus caused the fistula. He quotes Ewald, to the effect that fifty per cent of all the esophagotracheal fistulas are due to cancer of the esophagus. Among other causes, he mentions mediastinal tumors, tuberculosis, gumma and traumatism.

The case of Mouisset and Mangini[13] is of interest because of the valuable aid given by tracheoscopy. In this case there was difficult breathing, but a useless tracheotomy was avoided by finding the cause through the employment of the tracheoscope.

Basch[14] reports a case in which the patient had received an injection of salvarsan for gumma of the nasopharynx and pharynx. Twenty-four hours after the injection the patient died. The necropsy showed putrid bronchitis and gangrene of the lungs from tracheoesophageal fistula.

In a study of penetrating wounds of the neck, Henrichsen[15] showed that although the ordinary suicidal wounds are commonly made in the upper portion of the air and food passages, rare instances are found in which the trachea and esophagus are injured.

Moritz[16] relates the case of a woman, thirty-six years old, who died from a perforation between the esophagus and trachea. Tuberculosis was excluded as a cause, and although the evidence was not entirely conclusive, the author believes the lesion to be due to syphilis. Although the usual syphilitic ulcerative lesion results in the formation of cicatrices with stenosis, nevertheless syphilis may rarely produce a perforating ulcer.

Ponzio[17] reports the case of a man, sixty-three years of age, suffering from cancer. A fistula between the right bronchus and esophagus was diagnosed by the X-ray.

Schütze[18] made the diagnosis by means of the esophagoscope and X-ray. In this he was aided by the expectoration of colored frothy sputum after the patient had swallowed a solution

of methylen blue. The cause of the fistula in this case was doubtful. The question was between gumma and carcinoma.

Syphilis was probably the cause in a case reported by Dufour.[19] The patient was a woman, thirty-eight years of age. Death resulted from double bronchopneumonia, and the necropsy showed an ulceration upon the posterior wall of the trachea with fistula. Tuberculosis was excluded.

Pehu[20] relates the case of a bronchoesophageal fistula due to glandular involvement following tuberculous pericarditis.

An interesting case was reported by Martens[21] before the Society of the Charite Physicians, in which a patient, who had tuberculosis with empyema, had been operated for the latter. Later there developed an esophageal fistula opening in the back and ten bronchial fistulas. The patient remained in excellent general condition. The esophageal fistula required a tampon bandage every second day. Fortunately, there was no direct communication between the esophageal and the bronchial fistulas.

Woods[22] relates the case of a woman who had swallowed an artificial denture which had been allowed to remain in the esophagus for four months because of advanced pregnancy. A fistula was found from the gullet to the trachea. The denture was removed by esophagotomy. In the report of this case nothing was said of the subsequent history, although Woods says that the question now was how to treat the case, as the fistula still existed. In the discussion of this case, Dundas Grant thought that a large tracheal incision would be a good plan to reach the fistula, and called attention to a case reported in which a Hahn's sponge canula with a jacket on it of thin India rubber had been introduced and worn for a year, and in which the feeding could be done in comfort.

These cases were reported very briefly in the *Journal of Laryngology*, 1904, which accounts for this incomplete reference.

In 1905 de Navratil[23] gave the results of experiments upon dogs, in which he attempted an operative procedure for the closure of esophagotracheal fistula. He presented two operations, one of which gave very encouraging results and reminded one of the reference made by Dundas Grant in the discussion of Woods' case as given above.

A recent case reported by Beeler[24] shows the value of X-ray in the diagnosis. His patient died of pneumonia. The cause of the fistula was ascribed to syphilis. Unfortunately, no necropsy was obtained.

The case reported by White[25] is also instructive as showing what can be done by modern surgery. While White's case dealt with a fistula in the larynx, his method of procedure might be modified to meet the indications when the artificial opening is found lower down.

The case that I wish to report may be briefly stated as follows:

Mrs. P., aged twenty-four years, had suffered from severe asthma and bronchitis for a period of one year. There was a history of asthma in the grandfather, but no family history of tuberculosis. The patient gave no history of syphilis, being the mother of a healthy six-month-old child. She had had one miscarriage during an attack of measles. For seven months prior to her visit there was almost continual wheezing, shortness of breath, cough and expectoration. During the past year she had lost thirty-five pounds in weight. Six days before my examination the patient found it difficult to swallow both solid and liquid food. Every attempt caused violent choking and coughing. The difficulty in swallowing was sudden in its onset.

Upon examination an extremely emaciated woman with fever and rapid pulse gave one the impression of a patient suffering from tuberculosis; in fact, it was because of the suspicion that she was suffering from laryngeal tuberculosis that she was referred to me by Dr. Hoel of Fort Collins. Examination of the pharynx and larynx was entirely negative. Upon asking the patient to swallow a little water, a violent coughing attack supervened. Careful observation easily revealed the fact that the cough was produced after the water had passed the pharynx, a sufficient length of time elapsing from the moment the water was taken in the mouth to the time of the attack to give one the definite conviction that it had passed into the air passages after its entrance into the esophagus.

The physical examination of the patient by Dr. J. N. Hall showed moderate dullness at the right apex, with lessened voice and lessened respiratory murmur at times only. Abundant rales were found in the left lung, and there was rough breath-

ing everywhere. The abdomen was negative. There was no edema, no evidence of aneurism or syphilis, and no enlarged glands. The impression given Dr. Hall was also that of tuberculosis, although repeated examinations failed to reveal the presence of tubercle bacilli.

The diagnosis of perforation between the esophagus and trachea or bronchi was confirmed by the X-ray. A bismuth meal was given, and the radiogram taken immediately. The bismuth was seen passing from the esophagus to the trachea. (Figure 1.) After a few violent coughs another radiogram was taken, and the bismuth was seen to be scattered through numerous bronchi of the left lung. (Figure 2.) The extremely weak and hopeless condition of the patient, as well as the certainty of the diagnosis, obviated the necessity of a bronchoscopic examination. Death followed in twelve days.

The necropsy was performed by Dr. Hillkowitz, who reported as follows:

The thoracic viscera were removed en masse. There were no adhesions on either side to the pleural cavity. Pericardium, heart and valves showed nothing noteworthy.

Lungs.—The lower lobe of the right lung, its posterior aspect, showed three small abscesses the size of a pea. The rest of the visceral surface on both sides was perfectly smooth. On section both lungs showed numerous minute grayish spots sprinkled through their substance.

Esophagus.—This was split open throughout its length through its posterior wall. The mucous membrane was smooth and showed no congestion. At one point, the exact location of which is given later, there was a small perforation into the trachea. This aperture was round, with smooth rounded margins, and about one centimeter in diameter. On slitting the trachea through its anterior wall, the perforation was seen about two centimeters above the bifurcation, and in line with the left bronchus. The mucous membrane of the trachea was red, congested, rough and presented numerous elevations and depressions. In two or three places the depressions were about five millimeters in diameter, and presented a distinctly puckered appearance. These were doubtless scars. The mucous membrane in the neighborhood of the perforation was highly congested, the tissue disintegrating readily on touch.

Nothing noteworthy was seen in any other organ.

Microscopic Examination.—Sections from the lung showed the alveoli filled with desquamated epithelium, red blood corpuscles and leucocytes. There were small round cell infiltrations in the interstitial tissue, as well as around the bronchi.

Sections from the mucous membrane of the trachea at the site of the scars revealed a partial denudation of the epithelium and an extensive round cell infiltration of submucous and muscular tissue. The round cells were distributed everywhere, forcing their way between the muscular tissue fibers. The blood vessels were highly congested.

Neither in the sections from the trachea nor of the lung could tubercles or giant cells be demonstrated.

Pathologic Diagnosis.—Bronchopneumonia. Perforation between trachea and esophagus by severe ulcerative process in the trachea, the etiology of which is obscure.

The cause of the perforation in this case must remain in doubt. The history of the patient and the gross physical findings before death favor tuberculosis. This cannot be confirmed because of the negative laboratory and postmortem findings. The diagnosis of syphilis is negatived by the history of the case. Unfortunately, a Wassermann reaction was not made. Postmortem findings, as revealed by the healed ulcers in the trachea, the scar tissue and the final ulcerative perforation, speak strongly in favor of syphilis as the underlying cause.

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F[redacted]

FIGURE 1.

FIGURE 1.
FIGURE 2.

XXXVIII.

AN EPIDEMIC OF A SEVERE FORM OF ACUTE INFECTION OF THE THROAT, WITH ABSCESS FORMATION—REPORT OF FIFTY-EIGHT OPERATIONS.

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Albany.

During the past winter, particularly in December, January and part of February, the writer was fortunate in being able to observe an epidemic of acute infection of the throat occurring in Albany, and to a smaller extent in some of the neighboring cities. Altogether three hundred and eighty-four cases came under the writer's personal observation. Nearly all the cases were treated in Albany. A few were seen in consultation in Schenectady, Troy and Rensselaer. Some of the physicians in those cities stated that they had treated a large number of cases, but the writer was not able to secure any accurate data as to the exact number.

Of the total number of cases observed, three hundred and eighty-four, fifty-eight required surgical measures. Forty-four were cases of peritonsillar abscess, and in nearly all these cases diseased tonsils were present; in the majority of the submerged variety, with crypts high up in the upper lobe of the tonsil. Thirty-eight cases were unilateral, and in six, abscesses developed on both sides. Of the remaining fourteen cases requiring operation, in eight the abscess developed in the lateral columns of the pharynx, in two well up in the nasopharynx. There were two cases of infection of the epiglottis with great edema and some pus, two cases of abscess of the lingual tonsil, and two of retropharyngeal abscess, the last two occurring in children. It is worthy of note that in this series of fourteen cases, in ten there were no enlarged tonsils, and in the other four, healthy looking tonsils with no deep crypts. In the cases

then in which the infection did not originate in the tonsils, and in which no diseased tonsil tissue could be demonstrated, the infectious process selected other lymphoid structures like the lateral columns and the lingual tonsil.

We could not determine that the cases in this epidemic answered the typical description of epidemics of socalled "septic sore throat," nor could the streptococcus of Smith, which is supposed to be the organism responsible for septic sore throat, be isolated.

I suppose all cases of acute infection of the throat, with abscess formation, might properly be called septic, irrespective of the particular variety of streptococcus isolated in the cultures.

The writer has come to the conclusion that Semon's opinion, stated in his paper on the probable pathologic identity of the various forms of acute septic inflammation of the throat and neck, and since then in other papers, is the correct one.

Semon believes "that the various forms of acute septic inflammation of the throat and neck, hitherto considered as so many essentially different diseases, are in reality pathologically identical; that they merely represent degrees varying in virulence of one and the same process." It is known, for example, that a certain variety of the pneumococcus will produce localized edema. Welch, in his paper on the micrococcus lanceolatus, has determined by animal experimentation that this particular kind of pneumococcus or diplococcus, when injected subcutaneously, produced extensive local edema. This is a common observation also in cases of peritonsillar abscess and other forms of streptococcus angina, so that from a clinical and practical point of view it does not matter much what particular variety of the streptococcus is responsible for epidemic infections of the throat. The pathologic changes in the tissues are much the same.

Personally the writer is frank to admit that he has not been able to find the special distinctions and differences in the appearance of the throat, in epidemic sore throat, such as have been described by Smiley, who, working with Smith, investigated epidemics occurring in Boston, in which they isolated the particular variety of streptococcus now known as the streptococcus of Smith.

The epidemic in Albany was at its height during December and January; after the middle of February very few cases developed. During December and January the writer was called to see new cases daily. These occurred in all parts of Albany. We did not think that the epidemic was caused by infected milk, as the families afflicted got milk from many different dairies and milkmen. The cases of sore throat were distributed, as above stated, in all parts of the city, no greater number of cases occurring in families supplied by a particular dairy. During the worst of the epidemic so many new cases developed every day that we did not have time to study them carefully. The cultures that were taken in the beginning practically showed the streptococcus as the predominating organism. After that, as the cases in which no abscesses developed were so similar clinically, no cultures were taken, except in cases in which a distinct membrane was present, or when the diagnosis was in doubt. Patients were all very ill from the very onset of the infection, many of the cases starting with chills. In nearly all there was great prostration, and in the cases occurring in children, very high temperatures were the rule. Joint complications, acute arthritis and polyarthritis occurred in twelve of the cases, acute endocarditis in one case, and in twenty-four, examinations of the urine showed the presence of albumin and casts.

In the cases occurring among children between the ages of six and fourteen, eighteen in number, acute otitis media, requiring incision of the tympanic membrane, developed in eight. These cases were all clinically follicular tonsillitis, and could not be distinguished from the usual sporadic cases.

There were sixty other acute ear complications occurring among the cases during the epidemic. In the majority of cases the writer was called in time to incise the tympanic membrane very early, and this was undoubtedly the reason that no mastoid complications developed. There was only one mastoid, and that in a child, aged fourteen years. In this case the drum membrane had ruptured ten days before the writer was sent for.

It would make this paper much too long to report cases in detail, so all the cases of peritonsillar abscess will be grouped and considered as a whole.

The forty-four cases of this kind occurred in patients between the ages of ten and seventy-two years, the majority occurring in young adults betwen twenty and thirty years of age. There were four cases in children, three girls and one boy, aged twelve, ten, thirteen and fourteen years. In nearly all these cases, as before stated, the tonsils were found to be enlarged and abnormal in other respects. Thirty-eight cases were unilateral and six bilateral. A number of the patients between fifty and seventy years of age had not had any trouble with their throats for years, and came down with quinsy in this epidemic.

The abscess cases started very acutely, in many the usual symptoms being much intensified. Abscesses developed rapidly, with great edema of the surrounding parts, and swelling of the glands of the neck, particularly in the bilateral cases. In two of the bilateral cases there was great difficulty in breathing, due to an extension of the edema to the laryngeal mucosa. The constant use of an iced adrenalin spray afforded the greatest relief, and was continued after the abscesses were opened. Arthritis occurred in eight of the abscess cases, and endocarditis followed one case. There were four other cases of arthritis occurring in cases in which abscesses did not develop. Examinations of the urine were made only in the abscess cases, and in twenty-four albumin and casts were found.

In the management of the cases of peritonsillar abscess, I would like to mention a method that was employed with great success.

We have all experienced the difficulty in operating on these cases after they have been going on for a number of days, the patient at times not being able to separate the teeth far enough to admit even the handle of a teaspoon. If the cases are operated on early, this difficulty is done away with. Pus is present in the majority of the cases and can be reached after forty-eight hours.

For several years I have been following a method suggested by Ballenger—that is, a dissection backward toward the capsule until the pus is reached.

In fourteen of the cases operated on during the epidemic last winter, I have gone a step farther and have removed the upper lobe of the tonsil which had been loosened by the dis-

section. This leaves a large triangular opening for drainage. I have not found, and I agree with Barnes in this, that operating in this way during the acute stage, there was an increased chance for a more extensive infection. The entire sinus tonsillaris is usually infected in these cases, and patients so operated upon have done much better, and recovered more quickly than the ones treated by the usual incisions.

Barnes in his paper on the radical treatment of peritonsillar abscess by tonsillectomy, speaks of the difficulties of locating the pus when it is confined between the body of the tonsil and the superior constrictor muscle, and recommends dissecting out the tonsil in this class of cases. He has reported excellent results in ten cases.

It is unfortunate that we are frequently called to see cases of peritonsillar abscess so late, and when the edema on the affected side is so great that it is difficult and in some cases impossible to find the usual anatomic landmarks. In such cases the operative procedure described above was not attempted. It was performed only in selected cases.

The abscesses developing in the lateral pharyngeal columns are of particular interest, and are easily overlooked. The writer's attention was called to this class of cases particularly by Swain's paper on the subject. In a very instructive paper Swain reports several cases. In one, where he was able to make a thorough examination of the throat after the rupture of an unsuspected abscess of the lingual tonsil, a subsiding swelling of the epiglottis and aryepiglottic fold could be seen. The abscesses in the cases of this type occurring during the epidemic in Albany developed slowly, and for a number of days the writer could not determine the cause for the great distress and pain in swallowing that the patients complained of. In six of the cases the abscess was discovered high up in the lateral pharyngeal wall, the marked localized swelling finally calling my attention to the source of the trouble. In two other cases the soft palate was pushed forward. Pus was evacuated in all after the first deep incision. In the two retropharyngeal cases the bulging was found almost in the median line of the posterior pharyngeal wall.

Local anesthesia was employed in all cases, except in a few of the peritonsillar abscess cases in children, as the writer is of

the opinion that general anesthesia is not entirely safe in this class of cases.

Roe, in his very complete paper, "Phlegmons of the Upper Respiratory Tract," reported two fatal cases of abscess of the throat. In the first case, that of a young man, aged twenty years, a retropharyngeal abscess developed in the right side of the throat, which was opened. In spite of all efforts to save his life he died of exhaustion in three days. The throat infection was mixed—streptococci predominating. In the second case, a young woman, aged eighteen years, the throat was very much swollen, finally involving the laryngeal mucosa, and requiring tracheotomy. Patient died five days after the onset of the attack. This was also a mixed infection—pneumococcus and streptococcus.

Roe also mentions cases of faucial phlegmon in children resulting in retropharyngeal abscess. The abscesses were opened in all these cases, patients making good recoveries.

The larynx is frequently involved in acute pharyngeal infections, particularly when abscess formation occurs. The great edema of the surrounding tissues that frequently accompanies peritonsillar abscess and abscesses in other parts of the pharynx may extend to the laryngeal mucous membrane. Some of these cases are fatal.

Goodal has reported thirty-five cases of a very virulent form of ulcerative sore throat, twenty-five of which were fatal. The principal lesions were in the pharynx, but in nine the larynx was also involved. In these severe septic and ulcerative throat conditions the streptococcus is the organism most frequently demonstrated. Undoubtedly more virulent forms of streptococci are responsible for the serious complications, such as arthritis, endocarditis and nephritis, occurring with such throat conditions.

In the epidemic form which has been reported from many parts of this country and England the throat infection appears to be milder than in the sporadic cases.

In an epidemic in Baltimore, Hirschberg found, in addition to streptococci, pneumococci in forty-eight cases.

Davis and Rosenow, in the Chicago epidemic, found a peculiar streptococcus causing many serious complications and deaths. Cases had a sudden onset with severe prostration, en-

largement of the glands and other complications. The authors isolated an encapsulated hemolytic streptococcus of high virulency which sometimes coagulates milk.

Cases of mastitis were discovered in the suspected herds, and some cows that had not been suspected were discharging pus with the milk. This special streptococcus is thought to be related to the streptococcus pyogenes group which Leutscher has classified. He divided the group as follows: The ordinary streptococcus pyogenes group, which does not clot the milk or ferment raffinose. The streptococcus angiosus group, which often clots milk and ferments raffinose. He states that the streptococcus pyogenes group is usually associated with suppurations, erysipelas and septicemia, and that the streptococcus angiosus group is particularly associated with inflamed throats, and with malignant endocarditis and otitis.

A mild epidemic of sore throat occurred in Schenectady in 1912. Van der Bogart observed sixty-three cases, of which thirteen occurred in infants and thirteen between the ages of ten and fifteen years. It could be positively determined in this epidemic that the milk supply was not responsible in any way.

The ordinary run of the cases in the Albany epidemic, excepting the cases where abscesses formed, was characterized clinically by the intensely acute angina, great pain in swallowing, glandular swelling in many cases, extreme prostration and weakness, and unusually long continued temperature. The onset was usually very sharp, often starting with a chill and sharp temperature elevation. The writer was fortunate in having no deaths among his cases, although a number of the patients were seriously ill, and two just escaped tracheotomies.

In the treatment of the abscess cases after operation, patients received great relief from the use of frequent irrigations of the throat with normal salt solution and Seiler's solution. A fountain syringe was used, care being taken to control the flow so that a gentle current of fluid, under practically no pressure, was carried into the throat.

A study of the reported epidemics of septic and other forms of epidemic sore throat proves fairly conclusively that in the majority the milk supply was largely responsible. It is now generally known and believed that they are caused by a hemolysing type of streptococcus transmitted by the milk.

We can readily understand that milk as well as other body fluids may favor the development of a pathogenic power in the streptococcus.

In an epidemic at Rockville Centre, Long Island, milk was collected separately from each quarter of the udder of each cow. One cow showed mastitis, and streptococci isolated were bovine in origin. In one quarter with many streptococci the milk was flocculent. Both hemolyzing and nonhemolyzing strains were obtained, but few had the fermentative character of the bovine strain. The conclusion was reached in this epidemic that the cows were infected by the milkers.

Mellen, in his paper on the early diagnosis of streptococcic sore throat and the effects of vaccine in its treatment, comes to the following conclusions:

Early examination is valuable, as it gives a clue to the virulence of organisms.

It is not necessary to have symptoms for an individual to carry the organisms.

The acute cases respond quickly and decisively to a vaccine of proved immunizing power, if it is used early.

The condition should not go untreated, even though in most cases it is self limited, as it has many sequelæ.

Even after the symptoms are allayed, the body is still apt to absorb a few remaining organisms, with sometimes serious results.

Rosenow and Moon reported an epidemic of sore throat in Elmhurst, Illinois, occurring in February, 1914, which could be traced to the milk—isolated virulent streptococci from the milk.

Thirty cases used milk from the same dairy, which drew milk from eight farms.

Seven ounce samples of milk were centrifuged at high speed for twenty minutes. Sediment obtained was streaked in blood agar and inoculated into ascitis dextrose broth. Similar streptococci to the ones found in the patients' throats were isolated in nine out of eleven samples. They produced hemolysis on blood agar, and of them four were virulent for guinea pigs. On the use of pasteurized milk the epidemic subsided.

CONCLUSIONS.

1. The epidemic subsided when pasteurized milk was used.
2. Virulent streptococci showed preference for certain structures.
3. Organisms from milk resembled rheumatic strains culturally.
4. Involvement of muscles and joints occurred in patients infected by milk.

North, White and Avery, in a study of an epidemic of septic sore throat in Cortland and Homer, New York, in April, 1913, traced the source to milk. In three days a widespread epidemic developed. Mastitis was found among the herd in the dairy supplying most of the milk to both towns. The following results were obtained:

1. Seventy per cent of the cases in each town occurred among people taking milk from the same dairy.
2. Mastitis was found in two cows of this dairy herd.
3. Streptococci found in patients' throats were identical to those found in the milk from cows with mastitis.
4. The streptococci were similar to the ones found in cases of mastitis in cows of another herd.

At Finckley, England, in 1904, streptococci were demonstrated in throat cultures. In this epidemic five hundred and fifty cases were reported.

At Guildford, England, in 1903, streptococci were detected in the suspected milk.

At Christiania, in 1905, this was also found.

Mastitis in cows was noted in all four of these outbreaks, and the organisms were a hemolytic capsulated type. The cases appeared in winter and spring.

Symptoms.—Sudden onset, with sore throat, intense hyperemia and grayish exudate. Enlarged cervical glands with some suppuration was common. Extreme prostration was characteristic. The complications were otitis media, peritonsillar abscess, erysipelas, nephritis, endocarditis and myocarditis.

The infection was traced directly to the milk supply.

Streptococci were demonstrated in the mixed milk of the entire herd at a suspected farm.

Mastitis was found in the cows of this herd.

Streptococcic sore throat was diagnosed in the milkers.

Streptococcic sore throat was seen in other employees who handled the milk.

King has recently reported a series of cases of severe infection of the tonsils, followed by arthritis, endocarditis and other complications, in which the Conellan-King diplococcus was found in the crypts of the tonsils.

Seventy-eight cases have been observed by the author, in all of which this diplococcus was found.

In conclusion, I would like to give brief histories of two cases, one of abscess of the socalled lateral column of the pharynx, and the other an abscess of the lingual tonsil. Both occurred in young patients, aged eighteen and twenty-one years.

Case 1.—L. F., a young man, twenty-one years old. Was called to see him in December, 1915. He stated that for a couple of days he had had an extremely severe sore throat, starting with a chill and sharp temperature elevation. His physician, who called me to see the case, said he had been subject to sore throats. On inspection the entire pharyngeal mucous membrane proved to be intensely congested and reddened, the tonsils, however, not being particularly involved. Epiglottis and larynx appeared normal. I stated that it was an ordinary acute angina, and prescribed ice and an astringent gargle. His difficulty in swallowing got steadily worse, and I was again asked to see him at the end of another forty-eight hours. The throat looked much the same, except that high up on the right side of the lateral wall of the pharynx, above the arch of the soft palate, there was a decided bulging which pushed the soft palate forward. A deep free incision was followed by the escape of a rather thick pus. His recovery from this time was uneventful.

The second case, that of a young woman, eighteen years of age, started in a similar manner with a chill and temperature elevation. I was not asked to see this case until the throat symptoms had existed for five or six days. She was having some difficulty in breathing and great difficulty in swallowing. The right side of the fauces was very edematous, and examination with a laryngeal mirror disclosed the seat of the trouble at once. The lingual tonsil was enormously swollen and very edematous, so much so indeed that the larynx could not be

seen. This was of course responsible for her difficult breathing. The first attempt, a deep puncture failed to locate the pus. We then cocainized thoroughly and made a deep incision. This was followed by a very profuse hemorrhage and some pus. The bleeding was finally controlled by ice in the mouth and an ice bag externally.

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XXXIX.

ACUTE MASTOIDITIS WITH UNUSUAL SYMPTOMS INDICATIVE OF INTRACRANIAL INVOLVEMENT—OPERATION—RECOVERY.

By Edward Bradford Dench, M. D.,

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The patient was a young woman, aged seventeen years, who, following an attack of grippe, suffered from an acute otitis. She was seen by me on the eighth day after the inception of the middle ear inflammation. During this period two myringotomies had been performed. The night before I saw the patient the temperature had risen to over 103°, and on the day that I saw her the temperature was 104°. She presented the usual signs of a narrowed fundus of the external auditory meatus and a bulging drum membrane. The mastoid was exquisitely tender throughout. An immediate operation was performed. The mastoid cells were found to be filled with blood and some free pus. The mastoiditis was clearly of the hemorrhagic variety. A complete operation was done, and the dura in the middle fossa was exposed over an area about three-quarters of an inch square. The dura appeared healthy. The zygomatic cells were exceedingly well developed, and these were thoroughly eradicated. The upper portion of the wound was sutured and drained from its lower angle by means of two gauze drains. Owing to the high temperature, an immediate blood culture was made, and this was repeated on the second day. Both blood cultures were negative. The patient's temperature, at the time of operation, was 104°, and it fell with regular remission until about the fourth day after operation it became normal and so remained.

The patient left the hospital about ten days after the operation. She seemed very weak, and although she complained of no pain at this time she did not seem to be doing well. Two weeks and a half after the operation the patient began to have

headaches. The pain was located chiefly in the frontal and temporal regions. This pain was so severe as to keep her awake. Three weeks after the operation the patient came to the office with a temperature of 104° and a pulse of 142. She was immediately sent to the hospital and a blood culture taken. This was negative. The temperature gradually fell to normal, and the wound, which throughout had been doing poorly, seemed to improve.

The patient left the hospital again in about a week, and at this time had very little pain. One month and three days after the primary operation the headache returned and there appeared a paralysis of the external rectus oculi of the corresponding side. The patient at this time was suffering from severe frontal headache, was excessively tender to pressure over the first branch of the trigeminal nerve, was pale and anemic, and was exceedingly hysterical and lacrimose. Careful tests showed no ataxia, no nystagmus, fair hearing in the operated ear, a normal temperature and a normal pulse. Although the site of operation was the left side, there was no aphasia. The patient was given bromids, internally, to relieve the extreme nervousness from which she was suffering. These symptoms continued, with increasing severity, for about twelve days. During this time the abductor paralysis became absolute. The temperature and pulse during this period remained normal. The patient's general condition, however, steadily deteriorated. She became more and more nervous, the pain in the head increased, she lost flesh, and her progress was in every way unsatisfactory.

Six weeks after the first operation the patient again entered the hospital—on account of the severe headache and her general nervous symptoms. At this time the abducens paralysis was complete, but aside from this there was absolutely no evidence of any intracranial involvement. The hearing was fair on the affected side, there was no spontaneous nystagmus, and no evidence of any labyrinthine involvement. There was no aphasia, and an ophthalmoscopic examination showed both optic discs perfectly normal. Spinal fluid negative. The patient was seen in consultation with Dr. Pearce Bailey, who agreed with me that there were no signs of any intracerebral involvement. While the case resembled those cases first re-

ported by Gradenigo, I felt certain that there was no deeper focus of suppuration, owing to the thoroughness of the original operation. I could only explain the pain by assuming that there had been an inflammatory process extending along the dura from the area of original exposure inward to the Gasserian ganglion, and that this inflammatory process accounted for the pain. Such an inflammatory process would naturally account for the abducens paralysis.

One month and two weeks after the first operation I reopened the wound and extended the incision forward to a point one inch behind the margin of the orbit. The dura of the middle cranial fossa was exposed over an area about three inches in diameter. This area included the area of dural exposure at the primary operation and extended somewhat posteriorly to this; in other words, the dura of the entire middle fossa was exposed. The dura was carefully elevated by means of a flat spatula, and the entire floor of the middle fossa explored, including, of course, the roof of the petrous pyramid. Absolutely no pus was found. The dura at the site of original exposure was somewhat thickened, but otherwise the dura appeared to be perfectly normal. Firm dural adhesions at the anterior extremity of the first area of dural exposure were broken down by means of the blunt dissector. A folded rubber tissue drain was introduced to the very apex of the petrous pyramid. The anterior portion of the incision was closed and the mastoid wound was left wide open. I wish to emphasize here that absolutely no pus was found in either the mastoid wound or the middle cranial fossa. In other words, the first operation had been absolutely thorough, and this portion of the wound required no revision. The day after the operation the patient's temperature rose to 105°. She complained of no pain, however, and was perfectly rational.

From this time on she began to improve, and ultimately made a complete recovery. On the second or third day after the operation she developed beginning choked discs in both eyes. This swelling of the optic papillæ gradually increased and was more marked upon the operated side. The rubber tissue drain was removed from the middle fossa, and immediately the optic neuritis began to disappear.

The subsequent history of the patient is one of uninterrupted

recovery. The wound healed perfectly, the hearing on the affected side was excellent, and the abducens paralysis had practically disappeared when the patient was last seen. Subsequent reports of the patient show that she is in a practically normal condition.

Perhaps the most comprehensive article on this class of cases was written by Dr. Chas. E. Perkins, and published in the ANNALS OF OTOLOGY for September, 1910. The case which I have reported, however, presents some new points, and is particularly interesting from the fact that at the time of the second operation no extradural area of suppuration was found, neither was any deep focus of pus found within the bone, as so frequently occurs in these cases. In a number of cases reported the mastoid cells were found to extend into the apex of the petrous bone, and an evacuation of pus in this region was followed by immediate relief. In the present case the pain seemed to be due simply to a low grade of inflammation of the dura extending inward and involving the Gasserian ganglion. The sixth nerve was also involved as it crosses the apex of the petrous pyramid. The stripping up of the dura from the underlying bone seemed to cause a resolution of the inflammatory process with a subsidence of all the symptoms. The case is particularly interesting as bearing upon trigeminal neuralgia in cases of middle ear suppuration. The case certainly demonstrates that severe intracranial symptoms may occur without any demonstrable focus of pus in cases of this character.

XL.

THE ORAL CAVITY IN RELATION TO EAR, NOSE AND THROAT.*

By T. E. Carmody, M. D.,

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The oral cavity, located as it is at the beginning of the alimentary tract, and in the midst, it may be said, of the respiratory system, cannot but be most important pathologically, as it has been recognized to be physiologically.

Until recently the medical profession has ignored this very important cavity, leaving it entirely to the dentist, whom, while not considered competent, as he was said not to be a pathologist, was not only left in possession, but what he said or advised in individual cases was ignored or scoffed at.

The writer cannot, at this time, take up the matter of education of either the dentist or the physician, but suffice to say that he has long been of the opinion that the dentist should know more about the body in general, and the physician should know a great deal more about the oral cavity. However, under present conditions, they may each gain much by observation, not in the spirit of criticism of the other.

Upson expresses the feeling of the medical profession of a decade ago, as follows: "There seems to exist among physicians not only a disregard, but a distinct, though mild, dislike of the teeth as organs to be reckoned with medically; they being, as it were, an Ishmael not to be admitted to their pathologic birthright."

Lauder Brunton's essay on the subject is too little known and heeded, and few such systematic attempts have been made to correlate their disorders with the sufferings of the human race, except for the most obvious phenomena of pain. Ordi-

*Read before the Chicago Laryngological and Otological Society, February 29, 1916.

nary pain, at a distance, such as headache or neuralgia, due to the teeth, though well known, is commonly disregarded. Even the various reflex phenomena in children—convulsions, fretfulness and fever—are not now ascribed to irritation, either of teething or dental caries, but to digestive disorders. The state of recent opinion, as enshrined in epigram, is that the result of teething is nothing but teeth.

Although there has been some change in opinion in the last decade, there is yet too much tendency to laugh at what were the fears of earlier writers in medicine, and while we are able to disprove a few of their assertions, we must remember that we are as likely to make mistakes as they. In not keeping this in mind, we frequently go to the opposite extreme and miss the grain of truth, for such there is in the great majority of such writings. Furthermore, we hail with delight many new observations, new operations, etc., when they are simply stolen entire from other works.

The otolaryngologist has been interested for some time in the work of the orthodontist, on account of the change produced in the nasal cavity by the mechanical rearrangement of the teeth. It was the author's fortune to read probably the first paper on this subject presented to the medical profession, but unfortunately it was never published, but you are probably familiar with many of the other papers published on this subject.

There is nothing especially new in the line of reflex irritation of the nose, throat and ear, caused by pathologic conditions of the oral cavity, unless it is that produced by the condition known as pyorrhea, as this pathologic entity has been more recently considered by the medical profession from the standpoint of its effect upon, not only the gastrointestinal tract, but often the urogenital tract and ductless glands and joints, as well as the circulatory system.

In order to fully understand the reflexes, we must review our anatomy of the head, and especially that of the fifth nerve, with its connections through its ganglia with motor and

The relation of the oral cavity to the system in general has been given a great deal of attention by the physician in the last two years. Whether the infection always takes place through the gastrointestinal tract, as from the swallowing of pus, or

whether by absorption through the lymphatics surrounding the jaws, we must probably take the same view that we do in regard to the tonsils—that while a certain amount of the toxins may be introduced into the system in this way, the greater portion is discharged into the lymphatics or blood systems.

In a case of pyorrhea alveolaris we have a nearer approach to the tonsil than in any other condition in which the teeth are concerned, while abscesses in individual teeth resemble an abscess in any other portion of the body.

Whether the secretory function of the glands discharging into the oral cavity is affected by infection in that cavity more than by infection in other portions of the body, is a question that would probably be answered in the negative, with the exception of cases where inflammation, confined to these secretory organs themselves, is found.

Beck's formula of percentages of infection gives us something to work from, and we believe is correct in the early years of life:

From tonsils	20 per cent
From gastrointestinal tract	10 per cent
From appendix	10 per cent
From gall bladder and several other recesses	10 per cent
Total degree of infection	50 per cent
Resistance	50 per cent

These should be revised after the twentieth year, decreasing the percentage of infection from the tonsil by at least ten per cent, and increasing that of the gastrointestinal tract by the amount of decrease from the tonsil, the percentage of the increase in the gastrointestinal tract being credited to the teeth. It must be kept in mind that all infection from the mouth does not necessarily travel through the three routes above mentioned, but that in many cases, especially in the chronic mouth breather, infection of the larynx, trachea, and bronchi may take place in cases of aspiration of the infected material. However, these structures are probably saved from infection somewhat because of the fact that the mouth very soon becomes dry and the dried mucus on the tongue, lips and pharynx prevent the

bacteria from being aspirated, as they would be if the parts were kept continually moistened.

While the general surgeon and the internist are looking for the origin of diseases treated by them in remote parts of the body, and while many of these have been traced to the oral cavity, does it not behoove those of us who are working in the regions surrounding this cavity to examine it more closely for the origin of some obscure condition which we are called upon to treat? How easy it would be for the laryngologist (and many I am glad to say are availing themselves of the opportunity) to examine the teeth and jaws before looking deeper into the throat. How many of us would attempt to treat an ear condition without first making a thorough examination of the nose and nasopharynx? And this we have known must be done for some time, while at the same time looking through the oral cavity into the throat and only seeing the tonsil diseased, many of us, I am sorry to say, not knowing whether the teeth were present or not, with the exception, possibly, of the six anteriors. We could derive a great deal of benefit by having a dentist associated with us on the staff of each hospital; not the old time dentist, but the modern dentist, who knows not only how to fill and extract teeth, but who knows the pathology of conditions found in the oral cavity; who knows how to take skiagraphs and interpret same. Such a man should be of inestimable value to the laryngologist, and also to the internist and surgeon.

Few articles have been written on the subject of reflexes associating the mouth with the eye, ear, nose, or throat, although this subject is mentioned in many text books—attention being first called to it by Richter in 1795, and more practically by Hilton in his book on "Rest and Pain" in 1863, as well as in later additions. All books on oral surgery, first being that of Garretson, and two valuable articles, published since 1903; the first by Kate Wiley Baldwin in the *Journal of the American Medical Association,* January 31, 1903, and one by Clarence E. Ide, in the *Laryngoscope,* February, 1914; although many references have been made in other articles.

We are all familiar with the toothache, or pain in the jaw, which is frequently produced by impacted cerumen in the external auditory canal, and also with the cough produced when

an instrument causes pressure on the lower portion of the external auditory canal. The pain produced in the ear by ulcers on the tongue, abscessed condition in the region of the third molar or tonsil; the latter not always produced reflexly, but sometimes by direct pressure upon the eustachian tube. Ide treats this subject very thoroughly, by taking the nerves up under different heads, and we will follow him with a few slight changes.

A—Nerves of the external auditory canal and parotid gland.

1. Cervical plexus, branches of which are distributed to the hypoglossal, vagus, auricle, mastoid and parotid gland, besides branches to the sympathetic. The most important branch, auricularis magnus, arises from the second and third cervical nerves and winds around the posterior border of the sternomastoid, and after perforating the deep fascia ascends upon the muscle beneath the platysma to the parotid gland, where it divides into numerous branches.

The facial branches pass across the parotid and are distributed to the integument of the face; others penetrate the substance of the gland and communicate with the facial nerve.

The posterior or auricular branches ascend to supply the integument of the back part of the pinna, communicating with the auricular branches of the facial and pneumogastric nerves.

The mastoid branch joins the posterior auricular branch of the facial, and crossing the mastoid process is distributed to the integument behind the ear.

The occipitalis minor arises from the second cervical nerve; it curves round the posterior border of the sternomastoid above the preceding, and is directed almost vertically along the posterior border of that muscle to the back part of the side of the head. Near the cranium it perforates the deep fascia, and is continued upward along the side of the head behind the ear, supplying the integument and occipitofrontalis muscle, and communicating with the occipitalis major, auricularis magnus, and posterior auricular branch of the facial.

This nerve gives off an auricular branch which supplies the attolens auris and the integument of the upper and back part of the auricularis magnus. This branch is occasionally derived from the great occipital nerve. The occipitalis minor varies in size; it is occasionally double.

2. The auriculotemporal nerve arises by two roots, between which the middle meningeal artery passes. It runs backward beneath the external pterygoid muscle to the inner side of the neck of the condyle of the lower jaw. It turns upward with the temporal artery, between the external ear and the condyle of the jaw, under cover of the parotid gland, and, escaping from beneath this structure, ascends over the zygoma and divides into two temporal branches. The posterior temporal, the smaller of the two, is distributed to the upper part of the pinna and the neighboring tissues. The anterior temporal accompanies the temporal artery to the vertex of the skull and supplies the integument of the temporal region, communicating with the facial nerve and orbital branch of the superior maxillary.

The auriculotemporal nerve has branches of communication with the facial and otic ganglion. Those joining the facial nerve, usually two in number, pass forward from behind the neck of the condyle of the jaw to join this nerve at the posterior border of the masseter muscle. They form one of the principal branches of the communication between the facial and the fifth nerve. The filaments of communication with the otic ganglion are derived from the commencement of the auriculotemporal nerve.

It gives off auricular branches, two in number, inferior and superior. The inferior auricular arises behind the articulation of the jaw, and is distributed to the ear below the external meatus; other filaments twine round the internal maxillary artery and communicate with the sympathetic. The superior auricular arises in front of the external ear and supplies the integument covering the vagus and pinna.

Branches to the meatus auditorius, two in number, arise from the point of communication between the auriculotemporal and the facial nerves, and are distributed to the meatus.

A branch to the temporomaxillary articulation is usually derived from the auriculotemporal nerve.

Parotid branches, which supply the parotid gland, are also given off from this nerve.

The lingual or gustatory nerve supplies the papilla and mucous membrane of the tongue. It is deeply placed throughout the whole of its course. It lies at first beneath the external

pteryoid muscle, together with the inferior dental nerve, being placed to the inner side of the latter nerve, and is occasionally joined to it by a branch which crosses the internal maxillary artery. The chorda tympani also joins it at an acute angle in this situation. The nerve then passes between the internal pteryoid muscle and the inner side of the ramus of the jaw and crosses obliquely to the side of the tongue over the superior constrictor muscle of the pharynx, and between the styloglossus muscle and the deep part of the submaxillary gland; the nerve lastly runs along the side of the tongue to its apex (first running across Wharton's duct), lying immediately beneath the mucous membrane. (As it passes to the side of the base of the tongue it is accessible for operation.)

Its branches of communication are with the facial through the chorda tympani, the inferior dental and hypoglossal nerves, and the submaxillary ganglion. The branches to the submaxillary ganglion are two or three in number; those connected with the hypoglossal nerve form a plexus at the anterior margin of the hpoglossus muscle.

Its branches of distribution are few in number. They supply the mucous membrane of the mouth, the gums, the sublingual gland, the filiform and fungiform papilla, and mucous membrane of the tongue, the terminal filaments anastomosing at the tip of the tongue with the hypoglossal nerve.

The inferior dental is the largest of the three branches of the inferior maxillary nerve. It passes downward with the inferior dental artery, at first beneath the external pterygoid muscle, and then between the internal lateral ligament and the ramus of the jaw to the dental foramen.

B—Nerves of the tympanic membrane.

1. The outer surface of the drum membrane and anterior and inferior portions of the canal are supplied by the auriculotemporal branch of the inferior maxillary nerve.

2.—The inner surface of the drum membrane is supplied by the tympanic branch of glossopharyngeal.

C—Nerves and tympanum: Tympanic plexus (Jacobson's), consisting of:

1. Tympanic branch of ninth cranial nerve. From the petrous ganglion passes through the temporal bone between the carotid canal and jugular foramen; ascends to the tympanum,

entering by an aperture in the floor close to inner wall. Ends in two branches which ramify in grooves on the promontory.

2. Small deep petrosal nerve. From carotid plexus of sympathetic passes through wall of carotid canal to join Jacobson's nerve. Sends branch to great superficial petrosal through inner wall of tympanum in front of foramen ovale.

3. Small superficial petrosal nerve. From otic ganglion through foramen in middle fossa of skull (or through foramen ovale), passing backward to enter petrous bone again external to hiatus Fallopii; receives a filament from the facial nerve and joins Jacobson's nerve to assist in the formation of tympanic plexus. (The inferior maxillary branch of the fifth cranial nerve sends a branch to the tympani (tensor) muscle by way of the otic ganglion. The tympanic branch of the facial nerve supplies the stapedius muscle.)

D—Nerves of the eustachian tube: Tympanic plexus (See C).

E—Nerves of articulation and mandible:

1. Auriculotemporal branch of trifacial nerve, already described.

2. Masseteric branch of inferior maxillary nerve.

F—The otic ganglion is connected with the inferior maxillary nerve by its internal pterygoid branch through two or three short filaments, and it also receives a branch from the auriculotemporal nerve. Its sympathetic root comes from a plexus surrounding the middle meningeal artery. It also communicates with the glossopharyngeal and facial nerves through the small petrosal nerve continued from the tympanic plexus. Its branches of distribution are a filament to the tensor tympani and one to the tensor palati.

The submaxillary ganglion, situated in the deep portion of the submaxillary gland, near the posterior border of the mylohyoid muscle, being connected by filaments with the lower border of the gustatory nerve. The motor root comes from the corda tympani, and its sympathetic from a plexus around the facial artery. Its branches supply the submaxillary gland, Wharton's duct and the mucous membrane of the floor of the mouth.

G—Nerves of the nasal fossa. The sphenopalatine (Meckel's) is found in the sphenomaxillary fossa, close to the spheno-

palatine foramen. Its motor root is derived from the facial through the vidian. Its sensory root from the fifth and its sympathetic root from the carotid plexus through the vidian. Its branches are divided into four groups. The first or ascending group supplying the periosteum lining the orbit. It is also said to have branches to the optic nerve, cavernous sinus, and a communicating branch with both the sixth nerve and the ophthalmic ganglion. The branches of the second or descending group are distributed to the roof of the mouth, the soft palate, the tonsil and the lining membrane of the nose. The third or anterior group descends through the posterior palatine canal, passing forward, supplying the gums and communicates with the termination of the nasal palatine nerve. While in the canal it sends branches to the nose and maxillary sinus, and to both surfaces of the soft palate. Fourth, or internal branches are distributed to the septum and outer wall of the nasal fossæ.

Reflex pain from the mouth may deepnd upon an increase in number of teeth or the displacement of one or more of the normal number. The work of James and Pitts has changed our ideas to a certain extent as to the time when the permanent teeth should be erupted. As regards the temporary set, there is very little variation in number, although two teeth occasionally may be found fused, or an extra tooth may occasionally be found. With the permanent set we are much more likely to find supernumerary teeth. Bolk reported and exhibited about thirty specimens of additional permanent molars. In the Caucasian race we do not find supernumerary molars as frequently as they are found in the negro. However, we not infrequently find a supernumerary tooth in the anterior portion of the hard palate resembling a cuspid or lateral incisor. Whether any of these indicate a reversion to type or not, will not be entered into at this time. The conditions which most frequently confront us are due either to decay of the teeth, inflammation of the surrounding tissue, or abnormally placed teeth. A few cases will serve to illustrate.

Case 1.—Dr. W. B., aged twenty-seven years, consulted me in the year 1913, giving the following history: For about three years he has had severe neuralgic pains extending to the ear and over the side of the head, coming on at frequent intervals.

Ten days ago he consulted a dentist in his home city, who attempted to extract an upper right third molar, which was thought to be the cause. After one hour the operation was discontinued with the tooth loosened but still in place. On examination the tooth was found with one of the cusps broken off and somewhat loosened, and the patient informed us that his pains had been less frequent since the tooth was worked on. Under ether the bone was burred away and an exostosed tooth removed, since which time the patient has suffered no further pain, three years having elapsed.

Case 2.—Miss N., aged twenty-two years, consulted me in 1915 with her face very much swollen on the left side, giving the following history: About December 20, 1914, she noticed swelling of the face upon both sides, which she believed due to wisdom teeth, which were treated, but on account of the swelling the dentist hesitated to extract them. About January 10th they were extracted, but face continued to swell. Skiagraphs of face revealed nothing, but suppuration taking place, the abscess was opened on February 20th, and on examination of the discharge it was found to contain the ray fungus. This patient complained bitterly of pain in the ear and over the mastoid, but nothing ever developed in this region.

Case 3.—Mr. H. A., aged twenty-four years, a patient at the National Jewish Hospital. I was asked to see him on account of swelling in front of the ear and pain in the ear. He gave a history of receiving a blow upon the side of the head a few days previous. The ear was found to be perfectly normal, but a diagnosis was made of parotitis, which was subsequently changed to tuberculosis of the parotid gland. This case was reported in full in the *Laryngoscope*, October, 1914.

Case 4.—Mrs. G., aged thirty-five years, consulted me in December, 1914, complaining of pain in the right ear. The trouble was located in right lower third molar, which was extracted and pain disappeared. This tooth was badly impacted and pressing upon inferior dental nerve.

Case 5.—Mrs. S., aged fifty-two years, consulted me for what she believed to be cancer of the upper jaw and nose, as it had been under treatment for several months. On probing sinus found in the mouth, the probe encountered what apparently was the crown of a tooth, which was verified by X-ray.

Upon removal an opening was found into the floor of the nose, and the whole condition subsequently cleared up.

Case 6.—Miss H., aged seventeen years, consulted me on account of a swelling on the right side of her face, and the blocking of the right nasal passage. No pus could be found in the nose, and X-ray revealed a partially developed third molar in the right maxillary sinus, immediately above the first and second molars. A diagnosis of dentigerous cyst was made, and upon opening the sinus, a large amount of straw colored liquid was evacuated. The third molar was removed, and recovery was uneventful.

Case 7.—Dr. B., aged thirty-seven years, consulted me on February 20, 1913, complaining of a pain in the right ear. Examination of the ear showed it to be entirely normal, as was also the mouth. The following day, being unable to see me, he consulted another ear, nose and throat practitioner, who found that the patient not only complained of pain in the ear, but also of pain in the right maxillary sinus.

Case 8.—Mr. R., aged twenty-two years, dental student, consulted me in March, 1915, on account of a swelling of the neck over the larynx. On examination the neck was found very much swollen, extending up to the floor of the mouth. He gave a history of swelling at intervals, which would subside for a few days. On examination nothing was found in the larynx, and nothing in the floor of the mouth except a patulous foramen cecum. On passing a probe into the foramen cecum, it continued down the thyroglossal into the abscessed cavity. The swelling promptly subsided, and has not recurred.

XLI.

ADENOMA OF THE PALATE.

By Gordon B. New, M. D.,

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Neoplasms of the palate are commonly of a malignant nature, usually cancerous. They arise either in the palate itself or involve it from the nose, antrum, alveolar process or pharynx. The benign growths include papillomas, angiomas, fibromas, osteomas, odontomas, lipomas, mixed tumors, adenomas and other peculiar epithelial tumors. Adenomas of the palate are quite rare, although it seems hardly possible that they are as unusual as appears from a study of the literature. Possibly the rarity of case reports is due to the fact that the tumor usually shells out readily and often no microscopic examination is made. Moreover, some observers have classified adenomas with mixed tumors of the palate, although true adenoma is distinctly different and quite easily distinguishable microscopically. The term "adenoma of the palate" has been used loosely to indicate a slowly growing benign tumor, and tumors are at times reported as adenomas with no mention of the microscopic pathology of the growth.

Paget[1] reported thirty-one collected cases of clinical adenoma of the palate. He stated that the term adenoma in these cases is of clinical value only, implying nothing as to the microscopic appearance. In most of his cases there was no account of the microscopic pathology, and when present such accounts had little in common.

Since Paget's report (1886), I have been able to collect in the literature only eight cases with a pathologic diagnosis and five cases reported as adenoma without any microscopic pathologic diagnosis. This group seems far too small to represent the actual number of cases, but the factors already mentioned may account for the discrepancy.

Two cases of rare tumors of the palate reported herein, have been seen in the Mayo clinic; the first a case of true cyst adenoma, and the second an equally rare epithelial tumor, but not a true cyst adenoma.

Case 1 (157,265).—C. K., school girl, aged sixteen years. Examined December 6, 1915. Previous operation, tonsillectomy three years previously. Four days before examination a growth on the left side of the roof of the mouth was noticed; a physician was consulted, who punctured it, but did not take a specimen for diagnosis. He advised her to come immediately for treatment. It was not known how long the growth had been present; the girl believed that the left side of the roof of the mouth had always been larger than the right. There had been no other complaint; her general health was good, though she was very nervous. A tumor was found on the left side of the hard palate, two by one and one-half inches, extending into the soft palate. It was fairly regular in outline, and the mucous membrane covering it was normal except where it had been punctured. About this opening were signs of inflammation. The nose and sinuses were negative, and no glands were palpable in the neck. Wassermann negative. When taking a specimen from the tumor for diagnosis, about an ounce of straw-colored sticky fluid was evacuated. Microscopic examination of the specimen showed a cyst adenoma. (Figures 1, 2, 3 and 4.)

On December 13, 1915, the tumor was removed under ether anesthesia. It was found limited to the soft tissues of the palate, and did not involve the bone, although an excavation was noticed where it had rested. While the growth was readily dissected out, there was no definite encapsulation. The wound healed nicely and there is no recurrence to date.

Case 2 (156,093).—L. M. P., male, aged sixty-four years. Examined March 29, 1916. The man came to the Clinic because of a growth on the left side of the palate, noticed one year previously. The tumor did not seem to be increasing in size, and he had no inconvenience from it. Two weeks previously he consulted a physician, who lanced the tumor twice, but nothing was evacuated. No further treatment given. Except for this growth the patient was well. When examined, a tumor one and one-half by one inch in size was seen on the left side of

the palate at the juncture of the hard and soft palates. Mucous membrane normal over the tumor except where it had been incised by the home physician; tumor freely movable.

On March 3, 1916, the tumor was excised under local anesthesia. The growth was definitely encapsulated and easily shelled out. The wound healed readily. Microscopically the growth proved to be a peculiar epithelial tumor. (Figures 5, 6, 7, and 8.)

Adenomas of the palate may occur at any age. The youngest patient was a girl, aged sixteen years, reported in this paper, and the oldest a man aged sixty-five years, reported by Escat[2] in 1897. In the group of proved cases collected, including the one I have reported, were eight females and one male. The duration of the tumor may be from one year to fourteen years, but often the growth has been found accidentally on examining the mouth. Often in cases of the smaller tumors no symptoms are complained of, but the growth may increase to such size as to cause dyspnea and difficulty in swallowing. Dobson[3] reports a fibroma adenoma so large that it could not be removed between the teeth. The tumor may be situated in the hard or the soft palate or may involve both. Most frequently it is seen on the left side. It is not characteristic in appearance. The mucous membrane generally appears normal and unbroken, although Hutchison[4] reports two cases in which the surfaces were deeply ulcerated. In one case the ulceration resembled the crypts of a tonsil and contained cheesy material. In these two cases and Case 157,265 reported here, the tumors had diffuse margins with no definite capsule. In this respect they were quite different from those of other cases reported. Usually the growth is freely movable and rests in a saucer-like cavity in the bone like a dermoid. The tumor may be cystic, as that of Case 157,265, and the case reported by Marsden and White.[5] The outline and surface of the tumor are generally regular. No glandular enlargement was noted in any case reported. King,[6] however, reports a case in which the base of the tumor passed outward and backward behind the jaw and could be felt externally. In order to remove the tumor it was necessary to split the cheek. Dobson[3] reports a case in which the growth formed a swelling the size of a chestnut under the angle of the jaw. This swelling, however, we believe was an extension of the tumor rather than a gland.

HISTOGENESIS AND PATHOLOGY.

Paget[7] states that pathologically these tumors are embryonic in character and resemble closely tumors of the parotid which are manifestly embryonic. Morton[8] says the fact that in these tumors the same cells differentiate into gland epithelium and embryonic connective tissue cells of oval or spindle shape, proves the embryonic origin of the tumor from an early stage in the fetal development, and indicates a close analogy to the complex nature of the ordinary parotid tumor. MacCarty believes, however, that they are not of embryonic origin, but develop from the germinative layer of the epithelium of the palate. The normal basal layer of this epithelium may develop squamous cells or differentiate into glandular cells. Although these two tumors originate apparently in the same cell group, they are two distinct types. In the one the basal cells have grown down and differentiated into glandular cells, forming the picture seen in the true cyst adenoma. In the other the germinative layer of the epithelium has formed squamous cells and cell nests in its development. Areas are to be seen in which the differentiated cells are disappearing and others in which apparently they have dropped out entirely, the germinative layer alone remaining about the cavity. This theory as to the origin of these tumors seems by far the most plausible.

Most adenomas of the palate are encapsulated tumors, homogeneous on the surface and on section. Those reported by Marsden and White[5] and that in Case 157,265, however, were definitely cystic, containing fluid, According to Morton,[7] the characteristic features of an adenoma on microscopic examination are (1) cell nests, (2) adenoid tissue, (3) glandular structure, and (4) embryonic connective tissue. Hutchison[4] states that, in addition to these features, the tumors reported by him showed immense numbers of acini like those of the salivary gland and oral cavities filled with lymph cells. The latter possesses a regular fibrous wall similar to that of the lymphoid follicles of the tongue, tonsils, etc. It would seem that the term adenoma should be reserved for the type of tumor showing the characteristics of Case 157,265.

DIAGNOSIS AND TREATMENT.

In differentiating an adenoma from other types of tumors of the palate, it must be remembered that adenocarcinoma is not infrequently found in this region and also that sarcoma may be present. This must be taken into consideration particularly in the diagnosis of cases in which the tumor is found accidentally and the history necessary to eliminate malignancy is not obtainable. By far the safest plan is to excise a piece of tissue for examination. An operation may then be performed with a definite knowledge of the treatment indicated. When the tumors are small it is best to remove them under local anesthesia, as usually no difficulty is found in excising them. When large, however, such as those reported by Dobson[3] and King,[6] a more radical procedure may be required.

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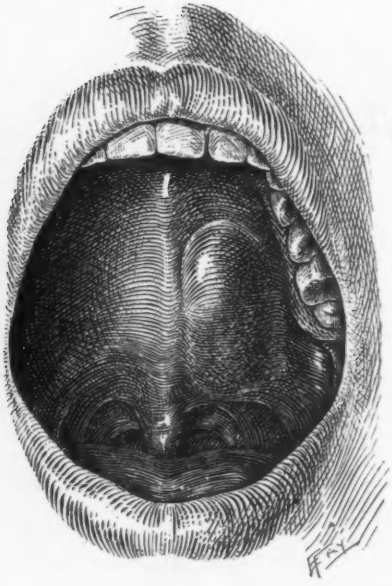

FIGURE 1. (157,265.)
Cyst adenoma of palate.

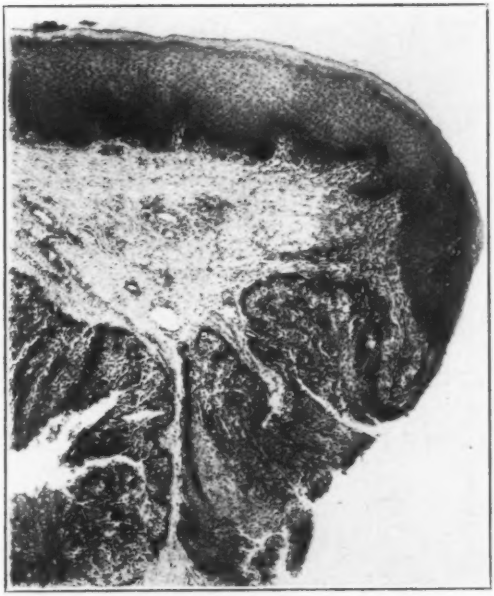

FIGURE 2. (Same as Figure 1.)
Downgrowth of the epithelium from the normal epithelium.

FIGURE 3. (Same as Figure 1.)
Glandular structure of the tumor.

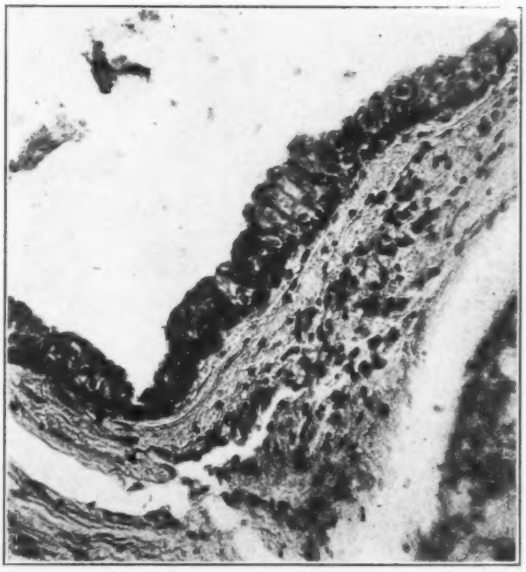

FIGURE 4. (Same as Figure 1.)
High magnification of the glandular cells.

FIGURE 5. (156,093.)
Unusual epithelial tumor of the palate.

FIGURE 6. (Same as Figure 5.)

Areas showing the cells' nests. In other areas the central part of the nests has dropped out, leaving the outer layer of cells.

FIGURE 7. (Same as Figure 5.)

Several areas where the central portion of the cell nests has dropped out.

FIGURE 8. (Same as Figure 5.)

Character of epithelium in remains of cell nests (high magnification).

XLII.

SURGICAL ANATOMY OF THE FRONTAL SINUS. DEMONSTRATION OF ANATOMIC PREPARATIONS.*

By George E. Shambaugh, M. D.,

Chicago.

Although the frontal sinus, because of its location, is the most exposed of any of the nasal accessory sinuses, it is the most difficult to treat of all of the sinuses when it becomes the seat of infection. The exposed position of the frontal sinus renders it the most accessible for external operation, but it is the one sinus where the external operation is least permissible. For obvious reasons the external operation for the relief of frontal sinus disease is restricted to those exceptional cases where an external opening has already taken place, or where intranasal methods of operation have failed to relieve symptoms of persistent severe pain resulting from the pressure of confined pus. This restriction of the cases where the external operation is indicated has become more and more generally accepted as it has become apparent that by more careful, painstaking intranasal work fewer and fewer of the cases of frontal sinus disease remain which cannot be cured or at least satisfactorily relieved by intranasal surgery. The objectionable scar in the most conspicuous part of the face, which persists even after the most favorable external operation, should constitute a contraindication for this method of operation except as a measure of last resort for the cases just indicated.

There can be no question that the intranasal operation for securing adequate drainage of the frontal sinus is more difficult and more dangerous than is the case with the other accessory sinuses. The difficulty and the danger of the intra-

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nasal operation lies in the anatomic variations existing in different frontal sinuses and in the relations of the nasal opening of this sinus to the cribriform plate of the ethmoid. For these reasons an examination of the surgical relations of the frontal sinus is of more than ordinary interest and importance. Because of the great variation in the size and shape of the frontal sinus, it is not feasible to take any one preparation and describe it as the typical frontal sinus. In this demonstration I shall attempt to show the more usual variations in the anatomy of the frontal sinus.

The first variation to which I shall refer is the absence of one or of both frontal sinuses. This is not an unusual variation. I have perhaps a dozen preparations showing an absence of one or of both frontal sinuses. This variation is, as a rule, bilateral. Occasionally there is a small frontal sinus on one side and an absence of a sinus on the opposite side. I have never encountered a large frontal sinus on one side with an absence of a sinus on the opposite side.

It is not always easy to make a distinction between a small frontal sinus and an anterior ethmoid cell, and such a distinction is often more or less arbitrary. The frontal, like the maxillary sinus, is an outgrowth from the ethmoid, and in a broad sense may be considered as an ethmoid cell. It is only when the cell is pushed upwards between the perpendicular plates of the frontal bone that the term frontal sinus seems applicable. Still, every ethmoid cell that has grown upwards between the plates of the frontal bone can hardly be called a frontal sinus. It is not at all uncommon for an anterior ethmoid cell to develop upward in this way within a well formed frontal sinus. Such a condition is not referred to as a double or a secondary frontal sinus. The ethmoid cell developing inside of a frontal sinus is still referred to as an ethmoid cell, under the name of bulla frontalis.

A well developed frontal sinus may exist where there is scarcely any extension upwards between the perpendicular plates of the frontal bone, but where the sinus lies entirely above the orbit.

In the most typical frontal sinus the development has taken place as an outgrowth from the anterior end of the infundibulum. In such cases the frontal sinus drains into the anterior

end of the infundibulum. It is not at all unusual for the anterior end of the infundibulum to open into a small or large ethmoid cell, while the frontal sinus has developed as an outgrowth from that part of the ethmoid just mesial to the infundibulum. It is seen, therefore, that a distinction between the frontal sinus and an ethmoid cell can be based neither upon the location nor yet upon the opening into the nose. The distinction is often quite arbitrary.

Contrasting with these preparations showing a diminutive frontal sinus or a complete absence of the sinus, are the preparations showing an unusually large sinus. Such sinuses are of special practical interest for two reasons. They are especially likely to be the seat of severe symptoms when they become infected, and should an external operation be resorted to they are more certain to cause a very disfiguring scar.

In expanding the frontal sinus may enlarge in three directions: laterally until it reaches the outer angle of the orbit, posteriorly over the roof of the orbit, extending even to the posterior part of the orbit, and upwards between the perpendicular plates of the frontal bone, extending in exceptional cases almost to the margin of the hair. An enlargement of the frontal sinus outward and upward can often be detected from the fullness above the eyes. In these cases the transillumination test often gives a very good idea of the anatomic condition, while the skiagraph gives a very accurate outline of the exact size of the sinus. The depth of the frontal sinus in its perpendicular enlargement is of special interest, since the degree of depression resulting from an external operation is largely dependent on this factor. This depth of the sinus is always greatest when the outlines of the sinus are expanded. I have preparations showing the separation of a full inch between the outer and inner tables of the frontal bone near the floor of a large frontal sinus.

Partial partitions within the frontal sinus often exist. They usually extend downward from the upper margin, thus separating the upper part of the sinus into several pockets. In other preparations such partition plates are found dividing the horizontal extension of the sinus laterally above the brow, as well as posteriorly above the orbit into several chambers. These partition plates are placed so as to interfere very little,

if any, with the drainage through the natural opening of the sinus, but their presence may interfere materially with the thorough exenteration of pathologic conditions within the sinus, even when performing the external operation.

The relations of the normal opening of the frontal sinus are of extreme interest in connection with the intranasal operation. They are by no means constant, but vary widely in different cases. As an unusual anatomic variation I have a single preparation in which the two frontal sinuses open into the nose through a single nasofrontal duct. The frontal sinus on the side where the nasofrontal duct is absent opens through a defect in the septum into the opposite frontal sinus. The one frontal sinus in this case has developed as an outgrowth from the other frontal sinus. The relations which are of particular interest are, first, those about the nasal opening of the nasofrontal duct; second, those of the ostium to the floor of the frontal sinus; and third, the relations of the ostium to the cribriform plate.

As regards the relations about the nasal opening of the ductus frontalis, it is important to keep in mind the fact that while the anterior end of the infundibulum opens typically into the nasofrontal duct, there are many cases where the opening lies to the mesial side of the infundibulum. In passing a canula into the frontal sinus it is sometimes necessary to introduce the point of the canula well back in the region of the bulla before it can be passed upwards and forced into the frontal sinus. In other cases the point of the canula must be placed far forward in order to allow it to pass into the sinus. It is sometimes necessary to use considerable pressure before a probe can be introduced into the frontal sinus. Such pressure is permissible only when the operator is perfectly familiar with the anatomy of this region and provided one adhere strictly to the following directions: The point of the probe must be kept away from the region of the cribriform plate—that is, it must be kept as far away from the septum as possible. The point must be directed more outward toward the orbit than inward. The point of the probe should be bent forward in a long curve, and should be directed at a right angle to the handle of the probe. This is necessary in order that when making pressure it will be possible to force the point of the probe for-

ward and outward and only slightly upward. By following these directions it will be possible even to force the point of the probe through the fragile walls of an ethmoid cell into the frontal sinus in exceptional cases where the probe cannot be made to enter through the natural opening.

The relation of the ostium to the floor of the sinus is best shown by making a horizontal section about the level of the supraorbital ridge. The ostium is found, as a rule, well back close under the internal plate of the frontal bone. In cases where there is a supraorbital extension of the frontal sinus, the ostium may be placed as far back as the middle of the cribriform plate. The relation of the ostium to the floor of the frontal sinus is determined largely by the surrounding ethmoid cells. For example, an ethmoid cell developing in front of the unciform process may extend upward in such a manner as to push the ostium well back. In exceptional cases only does an ethmoid cell develop in such a manner as to push the ostium away from the posterior wall of the sinus.

XLIII.

REPORT OF A CASE OF ACUTE MASTOIDITIS WITH INFLUENZAL MENINGITIS. TREATMENT BY OPERATION ON THE MASTOID AND ANTI-INFLUENZAL SERUM, FOLLOWED BY BRAIN ABSCESS — OPERATION — RECOVERY.

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Philadelphia.

E. C., eleven years old, was taken ill on Wednesday, October 2, 1913, on which day she came home from school, having been exposed to a severe wetting in a rain storm. That night she had a chill, followed by fever. A few days later she developed a pain in her right ear, which was followed by vomiting and headache. I was called in to see her, in consultation, by Dr. Knipe, the family physician, on the 12th of October, ten days after her initial chill. She complained of severe headache. Her temperature was 102°, pulse 120, and she seemed very irritable and had some photophobia. I incised her right ear drum and got a great quantity of pus from the right middle ear.

The next day, October 13th, she was admitted to the Pennsylvania Hospital, and I opened her right mastoid. Before the operation she was seen by Dr. Arthur Newlin, who agreed with me that she was suffering from meningitis, with acute mastoiditis. The child lay in bed, on the left side, with head retracted, crying out sharply from time to time. The pupils were rather large, reacted to light, and were equal. Her heart and lungs were negative. Kernig's sign was present to a slight degree. Her blood count showed 26,000 leucocytes, ninety per cent of which were polymorphonuclears.

Upon opening the right mastoid, which I did under ether, the process was found to be extremely cellular, and the bone very soft in consistence, a small amount of pus in the antrum

and in the cells towards the tip. Whilst she was under ether a lumbar puncture was made in the fourth interspace by Dr. Billings, and twenty cubic centimeters of spinal fluid, under great pressure, was removed. The patient reacted quietly after the operation. Owing to the appearance of the spinal fluid and the history of the case, it was decided to give the patient the antiinfluenzal serum of Dr. Flexner, of the Rockefeller Institute, of which Dr. Richardson had some in the Ayer Clinical Laboratory. This was done the same evening, and fifteen cubic centimeters of the spinal fluid were withdrawn and thirty cubic centimeters of the antiinfluenzal serum were injected under ethylchlorid anesthesia.

On October 14th the following report was received from Dr. Richardson of the Ayer Clinical Laboratory of the Pennsylvania Hospital:

"The spinal fluid presented the typical picture of that from a case of acute meningitis, being very turbid. Noguchi's butyric acid test was positive, and Fehling's copper solution was not reduced. Microscopic examination showed many pus cells, almost all of which were polynuclears. There were present some organisms which resembled B. influenzæ. Some of the sediment was cultured on human blood agar. After twenty-four hours there appeared several fine white colonies, made up of a small gram negative bacillus. These on further study proved to be B. influenzæ."

The same day, October 14th, another lumbar puncture was made by Dr. Billings under ethylchlorid anesthesia, and thirty cubic centimeters of spinal fluid were withdrawn and thirty cubic centimeters of the antiinfluenzal serum injected.

On October 15th the patient was very much more comfortable, still had some photophobia, and Kernig's sign was marked in the right leg, with much less rigidity of the neck, and the child was brighter. She was again given ethylchlorid and about thirty cubic centimeters of spinal fluid were withdrawn by lumbar puncture from the spinal cord, followed by the injection of thirty cubic centimeters of the antiinfluenzal serum.

On the 16th the child's condition was much the same, except that she was much more irritable. The lumbar puncture and the injection of the antiinfluenzal serum was repeated, but

very little fluid was withdrawn from the spinal column, and it was practically clear. Thirty cubic centimeters of the anti-influenzal serum were injected.

On October 17th the child's condition showed most distinct improvement. She was cheerful and bright, with no rigidity of the neck, and Kernig's sign had practically disappeared. There was still some twitching of the muscles in certain parts of the body during sleep. This was the date of the last injection that the child received.

Dr. Richardson furnished me with the accompanying laboratory note as to his findings throughout the period in which the child received injections of the antiinfluenzal serum.

"The further examination of fluid during treatment with the serum showed a gradually improving condition. There were no organisms found after the first specimen. The pus cells decreased in number, and the butyric acid and Fehling's tests later became negative. Several specimens of spinal fluid were examiner. The last showed an almost normal fluid with only slightly increased number of white cells. The butyric acid test showed only the smallest possible flocculent precipitate, while Fehling's solution was reduced."

Up to this date the pus from the ear discharge showed a pure culture of staphylococcus aureus.

From this date, October 17th, which was four days subsequent to the operation and to the first injection of the anti-influenzal serum, the child's case followed an uneventful course, until October 29, 1913. Her mastoid wound had practically healed, there being only a small surface on the lower portion which had not closed over. There was a slight amount of serous discharge from the external auditory canal.

On October 29th the child developed a slight temperature (100°), accompanied by some rapidity of the pulse. She complained of headache, and seemed very irritable. She had no Kernig's sign and no rigidity of the neck. Two days later, November 1st, she had several vomiting spells of a projectile character, and also seemed somewhat drowsy. Dr. William T. Shoemaker examined the eye grounds, and reported that the right optic disc was very hazy. Dr. Robert G. Le Conte saw the child in consultation with Dr. Arthur Newlin and myself. It was decided that the symptoms were indicative of

brain abscess, and on November 3, 1913, Dr. Le Conte and myself opened the cranium. On exposing the dura it was found to be very tense and bulging, and a large abscess cavity was found in the substance of the temporosphenoidal lobe. In the course of the operation the lateral sinus was accidentally punctured, but the hemorrhage was controlled without trouble. A rubber drainage tube was inserted into the wound, which was packed with plain gauze.

The subsequent course of the case affords nothing of any particular interest. Drainage of the wound was done away with on November 11th, eight days subsequent to the operation, and the patient was discharged, cured, from the hospital on December 7, 1913.

Dr. Richardson's report on the pus from the brain abscess showed a pure culture of streptococcus.

I have reported this case because it seems to me that the primary meningitis was undoubtedly influenzal and that the antiinfluenzal serum proved efficacious in its relief. The results following its administration were so striking that they seemed to be conclusive. I think it is also interesting to note that the laboratory report showed that the pus from the ear discharge was a pure culture of staphylococcus; the culture from the spinal cord showed the influenzal bacillus; and the pus from the brain abscess showed a pure culture of streptococcus.

XLIV.

SUPPURATION OF THE ACCESSORY NASAL SINUSES AS A POSSIBLE ETIOLOGIC FACTOR IN MULTIPLE SCLEROSIS.

By H. H. Stark, M. D.,

El Paso.

Having had occasion in the last year to look up the literature on blindness due to suppuration of the accessory nasal sinuses,[1] I was impressed with the similarity of the eye symptoms in this disease and those of multiple sclerosis, this being particularly true in those cases of sclerosis in which the diagnosis is made on the early eye symptoms, which may be the first and only symptoms of the disease in many instances.

This latter fact is emphasized by Bruns and Stoelting,[2] who, in the tabulation of their cases, show thirty-two per cent with ocular affections preceding the other symptoms for a variable length of time. This percentage, by different figuring, could be raised to fifty-eight per cent.

In the study of one case of sinus blindness this similarity is not so marked as where the symptoms are tabulated in the order of their frequency, as it is seldom we find all of them present in one case, diagnosis usually being made by the finding of one or two symptoms, and in some cases the presence of only one.

The time allowed for my paper was too short to go into this subject, and I only mentioned it in my closing discussion, in which I said: "I was waiting for someone to bring up the question of multiple sclerosis. The eye symptoms of this disease are so much like the cases I have reported, that I feel it quite possible for the nasal sinus suppuration to be an etiologic factor in sclerosis of the brain and spinal cord." So far as I can discover, this was the first time this question was mentioned in literature. Since that time I have made a fairly good search of the literature, and have found many points to

strengthen my theory that not only are the symptoms alike, but the two conditions may be due to the same cause.

I, therefore, think that a paper on this subject may be acceptable to the profession, and may do good by stimulating investigation, as my future work on this line will necessarily be slow, on account of the lack of neurologic material.

The eye symptoms in multiple sclerosis are amblyopia, nerve involvement, muscle involvement, including nystagmus, change of the pupil and field of vision. As in sinus blindness, these symptoms are not all present in one case, the diagnosis frequently being made by the presence of one or more, and it is necessary in certain cases to exclude hysteric amblyopia in the diagnosis of both diseases, but this can usually be done by the history of the case, interlacing peripheral color fields, and other signs.

I will take up the eye symptoms in the order of their importance and frequency, so that the similarity of the two diseases can better be seen.

AMBLYOPIA.

Ramer[3] classes sudden blindness as the most important eye symptom of multiple sclerosis. It is of a type resembling retrobulbar neuritis, and all authors agree that it may be the first and only symptom for a number of years, and it is usually monocular.

Aurbach and Brandt[4] state that in eighty per cent of the cases the blindness is monocular and comes without prodroma, or after transitory headache or vertigo. They say that the general symptoms come on as late as fifteen years after the blindness, as the disease is frequently very slow in its development.

Bramwell[5] says that visual defects, which are frequent and characteristic, often appear at an early date. Temporary unilateral amblyopia, of sudden onset and often regarded as hysteric in origin, as it is unaccompanied by change in the fundus, may be the first symptom of the disease. Other authors agree that it may be the first symptom, and that it may be a transitory blindness.

These symptoms just described agree in every respect to the amblyopia of sinus suppuration. The sudden onset, without prodroma, possibly a headache or dizziness, in many cases

a normal fundus, and even the percentage of monocular cases is exactly the same, eighty per cent.

This may also be transitory, as had been shown by numerous authors. I recently reported a case[1] of a physician, whose onset was sudden, the patient being immediately relieved by a spontaneous discharge of a small amount of pus from the nose, on the affected side. One can readily see that with an ordinary patient this fact could be easily overlooked, had the attention of the patient not been called to it.

OPTIC NERVE.

Ramer[3] classes the involvement of the optic nerve as second in importance of the eye symptoms of multiple sclerosis.

Bramwell,[5] in speaking of the nerve changes, says there may be a pallor of the temporal side of the disc, or an incomplete pallor of the whole disc, a small number of cases having been observed with optic neuritis, never, in his experience, amounting to true papillitis.

Bruns and Stoelting[2] quoted Oppenheimer as having had a case of atrophy of the optic nerve, this being the only cerebral symptom for twenty years.

Aurbach and Brandt[4] say that the eye only a few times shows the presence of neuritis or papillitis, atrophy occurring in twelve per cent of the cases.

Optic nerve findings in sinus blindness can be any change from paleness of the temporal side of the disc, neuritis, even papillitis. Great emphasis is placed on the paleness of the temporal side by many authors, but we do not find the same percentage of change existing in the two diseases. In my compilation of eighty-eight cases from the literature, there were fifty per cent showing optic neuritis, ten per cent of which were to a degree amounting to choked disc, but the atrophy percentage is practically the same as in multiple sclerosis, being eleven per cent.

OCULAR MUSCLES.

Ramer[3] classes this as third in importance of the eye symptoms.

Bramwell,[5] in speaking of multiple sclerosis, says diplopia, with no visible impairment of the ocular movement, is an

early symptom for which the patient seeks advice, and was present in thirteen of thirty-one cases recently examined by him, showing an involvement in his cases of forty per cent. He says that this involvement is of so slight a degree as to escape detection, unless specifically looked for.

Uhthoff[5] says twenty per cent of the cases have muscular involvement.

Ramer[3] says the muscular disturbance consists of (1) paresis, (2) nystagmus, or twitching of the eye resembling nystagmus. He says they are transient, often passing quickly, seldom lasting.

In my tabulation of sinus blindness there was thirteen per cent of muscular involvement, most of these being paresis of a transitory nature, as they were relieved by operation or spontaneous evacuation of the affected sinus; but if these symptoms are the same as multiple sclerosis, it must be especially looked for. There may have been a great many more with muscular disturbances which were never detected. So far, I find no case of nystagmus reported in sinus blindness, however, this might be present through toxic irritation of the labyrinth, which may also be the cause in multiple sclerosis.

THE PUPIL.

Ramer[3] classes this as fourth in importance of the eye symptoms in multiple sclerosis. He says that complete reflex immobility, miosis of the pupil, occurs very seldom, with occasional mydriasis and somewhat reduced reaction to light and a difference in the size of the pupil is met with.

Change of the pupil in sinus blindness is a point on which stress has not been laid. Two of my cases[1] had sudden dilatation of the pupil, occurring with amblyopia, but the pupil may be normal in size and reaction. In my tabulation of sinus blindness, I find reported twenty per cent with dilated pupil, four and one-half per cent with contracted pupil.

FIELD OF VISION.

Bramwell[5] says that in multiple sclerosis a central scotoma —usually bilateral—which may be for color alone, is often found, if looked for. He quotes Uhthoff, who classifies six

different kinds of visual field: (1) Fifty per cent of the cases had central scotoma, usually bilateral and relative, with no change in the periphery. (2) Rarely peripheral contraction with scotoma. (3) A considerable percentage with irregular contraction. (4) Isolated cases with regular concentric contraction. (5) One case with ring scotoma. (6) One case with central scotoma, which cleared up and was followed by a contracted field.

Aurbach and Brandt[4] emphasize the importance of central scotoma for red and green.

In sinus blindness we have had reported fields of all classes mentioned. Early claims were that there was a peripheral contraction. Later emphasis was placed on central scotoma. In late years considerable attention has been paid to the enlargement of the blind spot. However, the latter is still a debatable question among the profession.

The majority of authors emphasize the importance of central color scotoma as a diagnostic point, Fuchs[6] reporting a case of sinus blindness with operation and recovery, in which diagnosis was made on the finding of a central color scotoma alone.

Birch-Hirschfeld[7] claims that the first symptom is central scotoma, which may be followed later by a peripheral contraction of the field.

My experience has been that the central scotoma for red and green is the most important; however, I believe that the field of vision in many cases depends on the anatomic construction of the orbit, the different fields being due to the difference in the applied pressure.

EXOPHTHALMOS.

So far, exophthalmos seems to be a symptom not considered in multiple sclerosis, but from the pathology there is no reason why it should not be present.

In sinus blindness it is mentioned in twenty per cent of the cases, but this no doubt was not all of the cases with this condition, as Elschnig[8] claims that it can be found in half of the cases, by examining with a Hertl exophthalmometer. So it is quite possible that this condition may have been overlooked in multiple sclerosis, as it certainly has in numerous cases of sinus blindness.

NERVOUS SYMPTOMS.

Aurbach and Brandt,[4] in speaking of the cases of multiple sclerosis beginning with eye symptoms, say that the nervous change is usually very slight, and must be looked for. They mention, among the many nervous symptoms, different sensations in the two halves of the face, and parethesis of the hands. Both of these symptoms are not infrequently found in known cases of sinus involvement, and no doubt these and other symptoms have been overlooked many times by the rhinologist, due to lack of careful investigation.

MENTAL SYMPTOMS.

Bramwell[5] says profound psychic alterations are exceptional. Even in fully developed cases there may be no mental disturbance, and he mentions emotional control being impaired. This condition is also given as one of the symptoms of pseudo-sclerosis.

In investigating this subject I was impressed with this point, as one of my cases had a symptom that was entirely overlooked in my previous history of the case. While operating on him the man laughed continuously. No significance was placed on this, but recently he has informed me that several times following the operation this has occurred. At one time he was awakened from a sound sleep, and continued laughing for as much as a half hour afterwards, the attacks coming on without assignable cause. It is true that this case may not be considered as one of uncomplicated sinus blindness, but clinically it worked out as such.

ETIOLOGY.

Bramwell[5] says the cause is most obscure. No factor common to a large proportion of the cases has been demonstrated. In a study of the different authors, one finds that almost every known cause has been suggested for multiple sclerosis, but with all authors there seems to run a line pointing to the possibility of infection as the causative factor.

Pierre Marie[9] says that sclerotic patches are due to medullary localization of general infection, basing his views on the

clinical dependence of multiple sclerosis on infectious diseases.

Oppenheimer[10] says that with the majority of the cases the disease originates in an unknown base, or is a sequel after injurious factors, such as infection, and reports six cases following influenza. Competent neurologists are agreed as to the occasional occurrence of the disease after exposure to severe cold, especially in connection with abrupt changes of temperature.

Bramwell,[5] in considering the infection theory, says that the cases which seem to follow infections have no doubt had other symptoms present before the occurrence of the infection, infection simply making them worse.

Schnitzler[12] observed a case of multiple sclerosis in a woman of fifty-three years, following one month after an attack of influenza.

Church and Peterson[13] say the most important etiologic factors are infections.

Aurbach and Brandt[4] say that the relation of multiple sclerosis to retrobulbar neuritis is interesting in so far as the last named affection, in the great majority of cases, in acute as well as in the chronic form, is etiologically referrable to various intoxications and infections, arousing the suspicion that multiple sclerosis is likewise based on some organized or unorganized poison, crediting the increased frequency of influenza in the last decade with the greater frequency of multiple sclerosis.

If we admit that infection has something to do with multiple sclerosis (and we seem to have sufficient evidence in the literature to warrant this belief), we have exactly the same factor that causes sinus blindness.

PATHOLOGY.

In multiple sclerosis we find postmortem sclerotic patches in the brain and nerves. Just how early in the disease these sclerotic patches occur we are unable to say. However, it would look as though the process was slow, as is illustrated by Oppenheimer's case,[10] in which the general symptoms were delayed for twenty years.

Ramer[3] says that it has not been decided that the change of the nerve element or those of connective tissues and of the

blood vessels are primary in the disease. He is inclined to the idea that the disease starts from the surrounding tissues and not from the nerve fibers, for the fact is established that the axis cylinders are spared for a long time in the foci of sclerosis in the optic sheath, and lose only their medullary sheath.

Parsons[11] says: "The pathologic anatomy of the optic nerve in insular sclerosis is characterized by a significant difference in detail from that in other diseases, differing in accord with those found in similar lesions in other parts of the central nervous system. It resembles tabetic atrophy in the absence of pronounced signs of inflammation; it differs from it in the prolonged escape in the great part of the axis cylinders, in spite of their denudation by destruction of the medullary sheath."

Of the vessels he says: "The blood vessels are greatly altered, often profoundly. When this condition is present, there is usually an increase in number and a widening of the lumen of the finer ones."

He also says: "The pathologic condition is suggestive of the presence of a circulating toxin as the cause of the disease, but there is at present no proof that such is the case."

It does not seem possible that transitory blindness, muscular, pupil and nerve involvement, could come from plaques found in the nerve, for if they did, these conditions would be permanent rather than temporary; so these plaques must be late in forming, making quite tenable Ramer's idea that the beginning is from without. The late formation of the plaques, no doubt, causes the permanent condition in the central nervous system.

The pathology of sinus blindness has not been thoroughly investigated, but we are sure that the trouble in the nerve comes from without, for it comes from the involved sinuses. This would give us the same condition found in the pathology of multiple sclerosis.

The process of sinus blindness is due, no doubt, to two factors, both causing pressure. First, a low grade cellulitis, set up by changes taking place in the orbit, causing a gradual pressure. Second, a sudden increased pressure in the orbit, caused by either partial or entire giving way of the bony wall, bringing sudden and direct pressure on the contents of the orbit.

When we consider that in these cases of sinus blindness, most of them have a closed or partially closed cavity, with no drainage through the natural openings into the nose, and realize that we have a condition of pus under pressure, which acts by a dialysis of the toxin, or, in certain instances, direct infection by the microorganism, we can readily see how this persistent poison will eventually change both the brain and the nerves. This would make Parson's theory[11] of a toxin in the blood of special significance. It is not necessary for this pus to be present in large quantities, as in a review of the literature on sinus blindness we find quite a number of cases where the contents of the affected sinus was very slight. The trouble is evidently due to a gradual absorption of the poison. The condition may have been latent for years and suddenly lighted up by an acute infection or other conditions. If our theory is true, this would explain Bramwell's idea that the condition had been present before the infection.

CONCLUSIONS.

Since starting the work on this paper a number of months ago, two cases have come to my notice which bear directly on this subject.

In July, 1914, while in correspondence with Dr. Frank Billings, of Chicago, he gave me the history of a case which was worked out by E. C. Rosenow. This case is not reported in the literature. He says: "During the last summer we had one patient in the Presbyterian Hospital who suffered from all the symptoms and signs of insular sclerosis, and showed infection about his throat. The tonsils were removed. From the cultures made from the tissues of the tonsils, streptococci were found which when injected into two dogs produced focal infection in the spinal cord and all of the symptoms of multiple sclerosis. After the removal of the boy's tonsils we have had word that he has improved very rapidly."

Recently E. A. Shumway[14] has reported a case of multiple sclerosis which is of great interest on account of its having gone through such competent hands. The patient noticed dimness of vision in one eye in 1907, at that time having vision of 6/22. This was diagnosed as sinus blindness due to suppuration of the posterior ethmoid cells on that side. Later vision

fell to 2/45. After operation on the nose, vision gradually improved to 6/12, but again fell to 6/22, where it has remained. At the end of a year he noticed weakness of the right hand. From that time on varying symptoms of disease of the central nervous system developed, the diagnosis of multiple sclerosis finally being rendered in 1914, being agreed to by several neurologists who had examined him.

Shumway[14] says the case could either have been produced by sinus empyema or have been the initial symptom of the later developing of insular sclerosis, but from the light of subsequent events, it seems more reasonable to assume that the central nervous disease was the more probable cause. However, in his conclusion he says: "The query arises in my mind whether the disease in the present instance might have been caused by absorption of toxin from the purulent foci in the ethmoidal cells. So far as I know, no such theory has ever been advanced, and I offer it with some hesitancy."

These two cases strengthen my belief that multiple sclerosis is due to focal infection, and if statistics as given are correct, that fifty per cent of the cases of this disease have eye symptoms, the natural supposition would be that these cases have their origin in the nasal sinuses. Possibly others without eye symptoms may have a like origin, but no doubt most of them came from other sources.

The eye symptoms of multiple sclerosis and sinus blindness, which have previously been considered different diseases, are to my mind the same, one being an advanced stage of the other.

Having no facilities for carrying on elaborate laboratory experiments, I leave this question to the future investigation of the profession, to either prove or disprove.

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NEW YORK ACADEMY OF MEDICINE, SECTION ON OTOLOGY.

Meeting of February 11, 1916.

Otitic Meningitis Complicating Scarlet Fever.

By Henry L. Lynah, M. D.

DISCUSSION.

Dr. H. L. Abramson: The meningitis department of the Bureau of Laboratories of the Department of Health was called in on the case March 20, 1915. We had received twenty cubic centimeters of turbid cerebrospinal fluid from the house physician of Willard Parker Hospital. Examination of this fluid brought out the following: A great increase in the cellular elements, ninety-five per cent being polymorphonuclears; organisms that looked like rather large Gram positive diplococci, and a Gram negative diplobacillus resembling the Friedländer bacillus; albumin, 3 plus; globulin, 3 plus; prompt reduction of Fehling's. We considered the organisms present in this fluid contaminations, due perhaps to unclean receptacle.

On first examination the child appeared to be in rather light stupor; slight anterior posterior rigidity of neck; a positive Macewen; pupils reacted promptly to light and accommodation; there were profuse purulent discharges from both ears; the knee jerks were exaggerated; no paralyses observed; no Brudzinski or contralateral; signs of involvement of right mastoid; temperature, 104 degrees; pulse, 160; respiration, 40. A lumbar puncture was done that afternoon and fifty cubic centimeters of turbid fiuid removed under considerable pressure. Examination of this fluid showed the identical findings of the previous fluid. On the possibility that what looked like a Gram positive diplococcus in smear may prove to be a streptococcus in culture, twenty-five cubic centimeters of antistreptococcus was inoculated intraspinally.

Cultures from both fluids showed a mixture of a Gram positive diphtheroid bacillus and a Gram negative diplobacillus. These two organisms were separated, the former proving out as a chromogenic diphtheroid bacillus, producing yellow brown pigment, and the latter as a Gram negative diplobacillus, resembling the Friedländer bacillus. The diphtheroid grew very rapidly on ordinary nutrient agar, but the Gram negative diplobacillus grew scantily on plain nutrient agar but more profusely on glucose ascitic agar. The latter organism produced moist, mucoid, grayish colonies. Indol was produced in peptone solution, and it fermented lactose and dextrose. However, we were never able to demonstrate a capsule, even through cultivation in a fluid serum media.

On March 21, 1915, patient seemed brighter. Had been operated upon by Dr. Lynah for the mastoiditis of the right side the day before. He evacuated considerable tenacious purulent material. Lumbar puncture was done and fifty cubic centimeters of slightly blood tinged fluid was withdrawn. The fluid was thoroughly mixed with the blood, and it is our opinion that the blood was derived from the operation wound. This would indicate that there was free access between the seat of the inflammation in the mastoid and the subdural space. Twenty cubic centimeters of antistreptococcus serum was again inoculated. This spinal fluid and all subsequent specimens were free of organisms, both in smear and culture. Ptosis of upper eyelid and dilatation and immobility of the pupil on the left side were observed.

Lumbar puncture was done on March 22, 1915, and fifty cubic centimeters of straw colored, slightly turbid fluid was withdrawn under increased pressure. The color was due to hemaglobin derived from broken down red blood cells. The examination showed diminished number of cells, ninety per cent of which were polynuclears. Smear and culture were negative for bacteria; the content of albumin and globulin was diminished to 2 plus each; Fehling's solution was reduced promptly. The patient appeared to be brighter mentally.

Lumbar punctures were done on March 24th, 25th and 27th. None of these showed presence of organisms either in smear or culture. The cells, albumin and globulin content diminished rapidly, these elements in the fluid of March 27th being as

follows: No increase in cells; albumin, 1 plus; globulin, a trace.

An autogenous vaccine of the two organisms was prepared and administered as follows: March 24th, 250 million; March 26th, 500 million; March 28th, 500 million; March 30th, 500 million.

Comment.—It is our opinion that the meningitis in this instance was localized to the comparatively small area of meninges adjacent to the inflammatory process in the mastoid, and, further, that the virulence of the organisms recovered from the spinal fluid was low grade.

That the process was localized is indicated first and foremost by the fact that the Fehling solution was reduced as in a normal fluid—that is, promptly from first to last. Had the process become generalized, it would have involved the sugar secreting mechanism, the choroid plexus, and by impairing its secretory function, would have caused a diminution or absence of the reducing substance in the fluid. The rapid decrease of the cells and albumin and globulin, too, points in the direction of the localization of the meningitis, for in the generalized meningococcic meningitis we find considerable increase in these elements for some time after the organisms have disappeared from the fluid.

The organisms were of low virulence and probably derived from the middle ear inflammation, as they are commonly found in purulent discharges from the ear. Their absence from the fluid after the operation was performed on the mastoid indicates that the readier drainage through the mastoid wound proved effective in clearing up this localized meningitis. Had the organisms been of a virulent character, there would no doubt have been an extension of the meningitis in spite of the operation.

The use of the antistreptococcus serum and autogenous vaccine deserves little or no credit for the favorable result of this case. The low virulence of the infection, the operation on the original focus and repeated lumbar punctures, account for the result.

Only one other case of meningitis due to the Friedländer bacillus has come to our attention. This was a case of cerebrospinal meningitis that had been treated with antimeningitis

serum with favorable results. Not long after the spinal fluid had cleared up, he again developed symptoms of meningitis. Lumbar puncture brought forth a very tenacious mucoid exudate in very small amount, from which was recovered a pure culture of the Friedländer bacillus. On account of the character of the exudate and difficulty in removing same, a laminectomy was advised and performed. The patient drained more freely through this and made good recovery.

Diphtheroid bacilli are not very commonly found in the cerebrospinal fluid. Harris and Wade found them in three cases. In one case the smear was negative, but after two days' growth, culture showed a Gram positive diphtheroid bacillus. In the second case, a purulent meningitis resulting from a staphylococcus infected spinabifida, presented a few colonies of a Gram positive diphtheroid bacillus among numerous staphylococcus colonies, the diphtheroid being undoubtedly a skin contamination. The third fluid was from an adult male with high fever and opisthotonos. Forty cubic centimeters of slightly turbid fluid were withdrawn. This showed many pus cells and numerous Gram positive organisms, which proved to be diphtheroid bacilli. They were present in the specimen of fluid taken twenty-four hours later, but subsequently rapidly disappeared. Case recovered about a week after first lumbar puncture.

Dr. Dench said that the case was a particularly interesting one, as a meningitis of otitic origin is usually considered hopeless. With rare exceptions they are hopeless when operated upon, and it is advantageous to learn that certain cases do get well where the labyrinth is not extirpated or anything done except to relieve the pressure by lumbar puncture. It seemed to him that the bacteriologic findings and the vaccine therapy were rather mixed, as if the bacteriologist was harking back to the old shotgun prescriptions of thirty or forty years ago. We are still rather hazy as to the value of both the specific sera and the vaccines in cases of this kind. As he understood it, in this case the vaccines had been prepared from cultures from the spinal fluid. The case got well; that was the chief point. That is what all want to achieve, and the medication was certainly successful in this instance. By collating a large number of cases of this kind, we may be able to arrive at a definite conclusion as to what will be of value. We have some

cases with a turbid cerebrospinal fluid and streptococci in the fluid, and on culture we find the organisms dead. The presence of any germ in the cerebrospinal fluid does not necessarily mean that it was the cause of the meningitis. That was why he had asked about the cultures, and whether they were made from a germ from the spinal fluid. In some way the cerebrospinal fluid may in certain cases kill the germ—the same as in some cases of brain abscess we find a sterile pus. We know that in certain of these cases of meningeal inflammation the otitic symptoms may be simply coincident. In other cases of undoubted otitic origin, the infective organism may die spontaneously and the cases recover. Only by collating a large number of these cases can any definite conclusions be reached. The report of Dr. Lynah's case was of great value on that account.

Patient Presented Five Years After Operation for Congenital Bilateral Microtia.

DR. JOHN RANDOLPH PAGE: The patient, a little boy, was only six weeks old when he first came under observation. Dr. Page watched the child carefully for two years before operating on the right ear. In studying the literature of the subject he had not found much encouragement to operate, three reasons for not operating being repeatedly advanced: First, that whenever there is a deformity of the auricle with atresia of the canal there is always coexistent such deformity of the labyrinth that hearing cannot be obtained. Second, that tuning fork and bone conduction tests are necessary to demonstrate hearing. Third, that it is impossible to secure an open meatus because of the cicatricial contraction which takes place after operation.

When this child was a baby, the mother was satisfied that he had some hearing. When he was two years old one could attract his attention by whistling on fingers. That was enough to demonstrate hearing, without tuning forks or bone conduction tests. On looking up the development of the ear, it was found that the essential organ of hearing develops from an entirely different origin from that of the auricle and external auditory canal.

The case was presented to show the meatuses. By examining the child it could be demonstrated that he did have con-

siderable hearing. The mother claimed that she could call him from one room to another, without speaking louder to him than to her other children. In operating, allowance was made for cicatricial contraction. On one side the meatus now easily admits a speculum of medium size, and on the other side the largest speculum can be introduced. The first operation was performed five years ago, and the second was done four years ago.

One of the first things done after the operation was to test the labyrinth with water, to see if the child had a positive caloric, and the reaction was almost immediate.

When presented, the cavities were well dermatized, though there was some moisture in one. Dr. Page said that he did not remember in which one the tube was closed.

The mother stated that the child had called her attention to some frogs that were croaking a long distance off, a few months after the last operation.

This case was reported with a rather full account of the literature on the subject before the American Otological Society in 1914. In this report an interesting conclusion of Alexander's was mentioned, reached after many autospies on cases of congenital deformities of the auditory tract—namely, that in the vast majority of such cases the deformity is limited to the conducting apparatus alone, or to the perceiving apparatus alone; practically never is the defect present in both at the same time. He makes exceptions only in cases of nonviable embryos in which deformities of all three divisions of the ear have been found, and says that in such cases the deformity is always associated with other grave abnormalities elsewhere in the head, heart, or vascular system.

Another point of surgical value mentioned is the interesting observation accredited to Alexander by Castex and Burruyer, that malformation of the labyrinth always affects the facial nerve and auditory at the same time, and that one can conclude from this that all malformation of the external ear coexistent with facial paralysis indicates alteration in the labyrinth.

An X-ray was taken of this case when the child was small, and it demonstrated a tympanum; and at the operation rudimentary ossicles were found. Dr. Page said he believed that the stapes would be found in all of these cases.

DISCUSSION.

DR. BRAUN congratulated Dr. Page upon the results obtained, and said that he would like to make a brief preliminary report and show some photographs of a case he himself had tested. Three years ago he had under his care a boy, five years of age, with a double congenital microtia. He had a slight amount of hearing if one shouted very loudly. He had not learned to speak. There was complete absence of the external auditory canal. There was a ridge which corresponded to the margin of the auricle. The left side was operated upon first, through an ordinary mastoid incision, which revealed a fairly normal mastoid. Dr. Braun said he was able to demonstrate a normal antrum, aditus, and attic. In the attic were seen a normal incus and malleus. The articulation of the jaw lay against the anterior surface of the mastoid. The tympanic cavity was normal, but there was no drum membrane. In place of the drum membrane there was a mass of fibrous tissue, against which the jaw lay. He did not do a radical because the middle ear was normal; but he opened up as widely as he could and left the ossicles in situ. A circular opening was made where the normal meatus should have been, and the small skin flap was turned into the cavity, and the mastoid incision sewed up. An effort was made to get the cavity to epidermatize, but without success, and after five or six weeks the cavity had almost completely filled up with granulations. It was curetted out, and a skin grafting was done. This was successful.

Some months later the second ear was operated upon, and the same conditions were found as in the left side.

The child has been going to a public school for a little over a year, has been advanced twice, and his teacher has no difficulty in making him hear. He hears a moderately loud conversational voice without any trouble.

DR. PAGE said that he had been greatly interested in Dr. Braun's case, and with Dr. Braun's permission had included it with some others in the published report of his case. He had not followed the procedure of opening only down to the aditus, which was done as early as 1837 by Michael Jaeger, who advocated opening the antrum and allowing the sound waves to pass through the aditus to the tympanum without invasion

of the latter. Dr. Duel, in the case of a girl ten or eleven years of age, had followed that method several years ago, and later placed her in Dr. Page's care for dressing, but it had not been possible for him to keep the opening patent, so in the case presented this evening he had decided to make as large an opening as possible, exposing the internal wall of the tympanum. In all of these cases the anterior wall of the bony canal is absent, and the articulation of the jaw encroaches on the mastoid and posterior canal wall. In Dr. Page's own case he had been able, by entering the mastoid far back, to make a large radical cavity. Alexander attributes the great number of failures in these operations to the fact that the operators do not realize the position of the jaw in these cases and try to open not through the mastoid, but in what they take to be the line of the external auditory canal, and consequently come into contact with the jaw, and possibly paralyze the facial nerve. Dr. Page said he believes that the safest procedure is to enter far back in the mastoid and expose the sinus and middle fossa; then having these landmarks, one can be guided accordingly and work from the aditus and horizontal semicircular canal outward and backward, and have the opening point inward and slightly forward to the tympanum.

Report of a Case of Serous Labyrinthitis.

DR. CHARLES E. PERKINS: Miss M. C., aged eighteen years, had a mastoid operation on her right ear six years ago. The posterior wound healed, but the ear continued to discharge, and for the last three years patient has complained of attacks of pain. Otoscopic examination late in July, 1915, showed the membrana tensa absent and the internal tympanic wall dermatized except over the posterior part in the region of the oval and round windows, where granulations in small amount were present. Granulation also protruded from beneath the posterior fold. Shrapnell's membrane was intact.

Functional tests showed hearing in the right ear for whisper, two feet; upper tone limit, 21,000; lower tone limit, 90 D. V. Left ear: low whisper, twelve feet; upper tone, 21,000; lower tone, 55 D. V. Bone conduction increased on right side. Caloric, positive on both sides. Rotation, positive—slightly less to the right side. No nystagmus, vertigo or nausea.

Radical operation was performed in Dr. Dench's service at

St. Luke's Hospital, with primary skin graft, on August 2, 1915. Vault and antrum filled with granulations which extended into the old mastoid cavity, which had partly reformed by bony growth from the periosteum.

The postoperative rise in temperature was not over 100°. Two days after operation there was slight vertigo; four days after, there was marked vertigo, nausea, and vomiting; nystagmus to the left—that is, to the healthy side.

The pledgets were removed from the wound. The graft was firmly adherent to the radical cavity except in the posterior part of the tympanum, where it was separated from the bone by the accumulation of blood. After removing the blood, the patient was able to hear the shouted voice. The tuning fork on the vertex was heard better in the unoperated ear.

The patient assumed the characteristic decubitus of destruction disharmony—that is, lay upon the side of the good ear, thus enabling her to look in the direction of the slow component of the nystagmus and minimize the vertigo. After two days the vertigo diminished rapidly and she was able to be up. Four days later, that is, eight days after the operation, examination showed hearing for shouted voice; caloric reaction sluggish, but positive; rotation produced nystagmus to the involved side for about twelve seconds. The patient continued to become dizzy upon stooping or turning around suddenly for about two months; but the nystagmus to the left, that is, toward the good ear, continued with scarcely noticeable diminution for five months, since which time it has been gradually disappearing.

The hearing in the right ear has returned to about the same as before the operation; the caloric reaction is positive; rotation produces a nystagmus to the right of about fifteen seconds, although the eye movements are sluggish.

In my experience it is unusual for nystagmus to persist in a marked degree for five months, after either a serous or a purulent labyrinthitis. If the static function of one labyrinth is totally and permanently ablated, as in diffuse suppurative labyrinthitis, the opposite labyrinth accustoms itself to the new order of things, and the symptoms of destructive disharmony, including the nystagmus, pass away within a month, as a rule. In this patient, the fact that the function of the static mechanism on the right side has been gradually return-

ing seems in some way to act in causing the nystagmus to the left to persist.

The process within the labyrinth was no doubt caused by inflammation or the accumulation of blood beneath the graft at the side of the oval and round windows. Labyrinthine complications are more common in patients who present granulations in the neighborhood of these fenestra; so before applying a graft in these cases one should be sure that the hemorrhage has ceased and the tympanum is dry.

Not infrequently there is nystagmus and vertigo during the first four days after the radical operation, and not due to any intralabyrinthine disease. One hesitates before removing the grafts in such cases, but the possibility that there may be some disease of the labyrinth makes one more or less uneasy.

It is a well known fact that deafness which gives tests characteristic of lesions of the perceptive mechanism is usually an early symptom in serous and purulent meningitis. I have been accustomed to rely upon the Weber test in determining this point. For instance, if the fork placed upon the vertex is lateralized to the operated ear, it is, to a certain extent, an indication that the perceptive mechanism is intact. The patient must be fairly intelligent. There are, no doubt, possibilities of error in this test, but the information afforded has seemed to be of value.

DISCUSSION.

Dr. Braun asked if the fistula test was made before the operation, to which Dr. Perkins replied in the negative. Dr. Braun then said that in many of the cases in which a serous labyrinthitis followed the radical mastoid operation, a labyrinthine fistula was present before operation.

Dr. Braun said that he disagreed with Dr. Perkins as to the value of the Weber test. This had proved a very unreliable test in his hands. In a number of instances, patients with a dead labyrinth referred the Weber to the diseased side. In a patient operated on by Dr. Richards several years ago, in which he did a complete labyrinthectomy, the patient lateralized the Weber, after operation, to the ear from which the cochlea had been removed.

Dr. Friesner emphasized what Dr. Braun had said about the Weber test. If one has a suspected labyrinthitis, and the

Weber test be referred to that side, one would conclude naturally that that ear has function. He recalled a case upon which Dr. McCullagh had operated, in which the cholesteatoma had completely destroyed the labyrinth. The patient was a very intelligent young man, and he referred the Weber definitely to the diseased ear. Dr. Friesner was misled by this, and believed at first that the diseased ear had function, but the findings showed that the labyrinth was completely destroyed. Furthermore, the other tests showed that the labyrinth had no function.

Dr. Dench said that he also would like to emphasize this point in cases where there was not a destructive labyrinthitis, but where the labyrinth was involved from a nonsuppurative process. He had seen patients who were absolutely deaf in one ear refer the fork to the deaf side. He had seen that himself, though he did not know the cause, or how to explain it. It certainly does occur in suppurative labyrinthitis and in cases where the labyrinth has been removed. It occurs also in the nonsuppurative labyrinthine cases.

Dr. Perkins said that he was not unaware of the uncertainty of the Weber test, and had not claimed for it infallibility. In the presence of deafness due to lesion of the perceptive mechanism, we do not expect increased bone conduction. The fact that deafness of this kind is an early symptom in both serous and purulent meningitis, justifies us in expecting a diminished bone conduction in the involved ear. That there are exceptions, real or apparent, to this rule will not be denied. Nevertheless, he did not feel that he had claimed too much for this bone conduction test. No doubt, the safest way would be to remove the dressing and test the hearing, using the noise apparatus on the opposite side.

A Contribution to the Pathology of Mastolditis With Reference to Thrombotic Conditions. Lantern Slide Demonstration.

Dr. Samuel J. Kopetzky said that he had not prepared a paper on this subject, but that he had been doing some work with the idea of coordinating the clinical findings with a study of the pathology of these cases, and that he presented these specimens at the invitation of the chairman.

DISCUSSION.

DR. MCCULLAGH said that this work which Dr. Kopetzky had been doing was most interesting and instructive. He had been especially interested in what had been said about the pathology of mastoiditis—the division of cases into the hemorrhagic and the coalescent types. He himself had felt that the latter was purely a secondary stage of the hemorrhagic condition which Dr. Kopetzky had described, and he believed that the hemorrhagic type does go on to this coalescent type in many cases. It would make the slides of more value if one could know the length of time that the disease had existed before the specimens were taken; it would give more light upon the time of change from the hemorrhagic to the coalescent type, if one merges into the other.

Paper: The Surgical Treatment of Persistent Suppuration of the Jugular Bulb.

BY JOHN MCCOY, M. D.

DISCUSSION.

DR. DENCH said that the members of the section were indebted to Dr. McCoy for his clear exposition of the various operations for the relief of thrombosis involving the jugular bulb. They had been very skillfully collated and presented. There were good points in each operation. As Dr. McCoy had stated, the position of the jugular bulb varies in different subjects. In some cases it is the same on both sides; in some cases the dome is high on one side and low on the other; in some it is away forward, in others away back; and in some there is no dome. These latter cases are easy to operate upon.

Dr. Dench recalled two cases where he had done the Gruenert operation. Both patients died, but the operations were not particularly difficult. In both instances the jugular vein had been excised previously for a jugular sinus thrombosis. In all cases he has made the mastoid incision continuous with the incision in the neck, for the ligation of the jugular vein. In one instance the vein had been taken out by an assistant, who had not taken up the vein as high as he himself was accustomed to doing. It was a simple matter to pick up the end of the

jugular vein in the neck in this case and follow it upward, then expose the sinus and work forward toward the bulb; then, by working far behind there was no danger of injuring the facial nerve. The facial nerve was cut in the first case. That was practically the only danger. Dr. Dench said that he almost always cuts the occipital artery in cases of jugular excision. He was in favor of a combination of the three methods in bulb cases. In the first place, make the mastoid incision continuous with the incision made in the neck for the jugular excision; then the vein and the nerve are exposed. It does not make much difference which technic is employed, for the technic adapted to one case might not be suitable to another, but by remembering the anatomic points the operation was freed of all difficulties.

All of his operations had been secondary to previous sinus and jugular operations. The primary cases must be very rare. He had followed one procedure in two or three cases. If one works very far forward and exposes the lateral sinus, in cases of sinus thrombosis, and follows on to the horizontal portion of the sinus to where the external wall becomes the inferior, it can be followed for a very considerable distance. In these three cases there was a considerable suppuration in the bulb, and it was easy to clean it out and pack it. The patients got well very rapidly. Dr. Dench said that where it was necessary to do the radical procedure he believed the best results could be obtained by a combination of the various methods presented by Dr. McCoy.

Dr. Whiting said that it has often occurred to him that the very elaborate methods of accomplishing results might be eliminated by doing a thorough operation in the beginning. In saying this he meant no reflection on the method of any operator, but he has performed a great many operations for sinus thrombosis and has never yet had a thrombosis of the bulb, although he has had a good many cases of thrombosis of the bulb in connection with operations by other men. As the sinus operation is commonly performed, the men content themselves with exposing an insufficient portion of the sinus. They expose the knee and the descending portion, and when they get fairly well down they speak of having carried the incision as far as the bulb. They are nowhere near the bulb, but only

where the descending part of the sinus moves around in approaching that part.

In his own operations, Dr. Whiting has always followed the procedure mentioned by Dr. Dench, exposing posteriorly a good part of the occipital bone; then, instead of cutting the groove in the dimensions which correspond to the concavity, going posteriorly and cutting away all the jugular portion of the occipital bone between the transverse process. Then the operator has a space in which he can follow forward the sigmoid sinus into the bulb. That is a modification of Gruenert's procedure, except that it is not necessary to subsequently open the neck, as Gruenert does. Dr. Whiting said that he has done this many times in operating after other men, and has never had any trouble in cleaning out the bulb. Gruenert's operation is a well recognized one, but Dr. Whiting has never found it necessary to perform it.

Dr. Whiting is convinced that if the primary operation is performed, removing a large part of the occipital bone well back to the jugular process, then following the curve of the jugular sinus toward the jugular foramen, the posterior and lateral border can be taken away and the jugular border will be open, and it will never be necessary to do the Gruenert operation.

Dr. Braun said that he had occasion to open the jugular bulb a year ago, and was surprised to find how easily it was opened from behind, exposing the sinus well down (demonstration on blackboard), and with a rongeur removing the wedge of bone between the lower part of the vertical limb of the sinus and the bulb, keeping to the medial side of this wedge, in order to avoid the facial nerve. It did not take more than five minutes to make an opening half an inch in diameter. As there was a good deal of inflammation around the vein in the neck, the neck incision was extended up to meet the mastoid incision, and a probe passed from the open end of the vein up to the bulb. The case recovered.

NEW YORK ACADEMY OF MEDICINE, SECTION ON OTOLOGY.

Meeting of March 10, 1916.

Entotic Aural Bruit, Audible on Auscultation of Head, Due to Tumor of the Pituitary Region.

DR. L. D. ALEXANDER: The patient, George D., was a negro, forty-two years of age, born in St. Kits, West Indies. He was married and had four children. Father was alive, eighty years of age; his mother died when about fifty (?); she had sixteen children.

Previous History.—Had measles at six years, no complications; had frequent headaches, right frontal and occipital regions, until twenty-one years of age. These disappeared when he left the West Indies. He denies venereal infection. Two years ago had a painful swelling of the left testicle, which subsided after rest and suspension. Has had hay fever every summer for the last fourteen years.

Present History.—Following his hay fever attack eighteen months ago, noticed a hissing sound in his right ear. This sound varies in intensity, and is especially noticeable when in a quiet place or upon exertion. He has had diplopia for the last year, and notices increasing difficulty of vision in the right eye; has daily headaches over the frontal and occipital areas, which are especially severe in the daytime.

Examination.—General physical condition, good. Post cervical glands, palpable. The chest and abdomen reflexes are negative.

The left ear is negative. Auscultation over the right ear reveals a blowing bruit, synchronous with pulse beats. The sound is heard directly over the entrance to the canal, and less distinctly forward and upward over the temporal region. The sound can be obliterated by firm pressure over the carotid.

The tubes are patent; tympanic membranes, normal; hearing, normal—Weber, Rinne, and forks.

Eyes are prominent. Pupils react to light and accommodation, but rather more slowly than normal. Media, clear; fundi, negative; optic disc and vessels appear normal; no

tension. Vision: right, 20/30 plus C plus 3.50 sph.; left, 20/20 minus C plus 3.50 sph. Fields generally contracted for form and colors. No scotomata. Has diplopia increasing to left; image level; diplopia is light and indicates a slight paresis of left rectus; motility good.

Pathologic Examination.—Urine, negative. Blood, negative. Wassermann, 4 plus. Blood pressure, 140.

Radiographs of chest and anterior view of sinuses are negative. The lateral view shows enlargement of the sella, with probable erosion of some of its walls.

Radiographer's Report.—Plates 3743, December 20, 1915; 3743b, February 21, 1916. "The sella measures fourteen millimeters long by thirteen millimeters deep, and the area (averaged from the four plates by means of the planimeter) is one hundred and forty-five square millimeters, which is considerably larger than it should be. The posterior clinoids are elongated by apparent erosion at the base. A tumor in this location would be in contact or very close to the internal carotid as it leaves the carotid canal in the petrous pyramid; or there might be an aneurism of the carotid, possibly both tumor and aneurism, which would account for the bruit.

Sphenoidal sinuses are large and clear.

(Signed) GEORGE S. DIXON, M. D."

DISCUSSION.

DR. CHARLES GRAEF said that he was especially interested in this case, as he had been the first to see it, the patient having been referred to him last year by a practitioner for tinnitus and diplopia. He had made a diagnosis of aneurism, but had not determined the exact location of the vessel involved. It would seem to him that if the X-rays were to be accepted here as decisive, the pituitary gland should be expected to show more evidence of disturbance after a period of involvement lasting over a year, as this one has. No localizing signs or symptoms were found outside the X-ray shadows, and in intracranial conditions there were not very trustworthy. While under his care, the Wassermann reaction being four plus, the patient was kept on mixed treatment and given a series of mercurial inunctions. Outside of that, he could think of nothing likely to be of value but salvarsan, and as that was very expensive and difficult to

obtain, the patient was not admitted to the hospital, and drifted into other hands.

He expressed his gratification at hearing the complete history given and at seeing the case here. He had never before heard of anything like this case. He had spoken of this patient to some of the members some weeks ago, and had intended to arrange for its presentation before the section.

DR. ALEXANDER in discussing the case stated that he had shown plates to Dr. Quimby, who agreed with findings of Dr. Dixon.

DR. EAGLETON said he believed the condition was due to an aneurism of the carotid which had widened the sella by pressure, and that he did not believe the pituitary involved.

Labyrinthitis Following Mumps.

DR. NORTON L. WILSON said that he presented this case not because of its rarity, but with the hope of either being able to do something for the restoration of the patient's hearing or to convince her that nothing could be done.

The patient, Marguerite F., was fifteen years of age, and had never had any trouble with her ears before the present attack. On December 25, 1915, she was taken ill with diphtheria; that same evening she was given four thousand units of antitoxin. The throat cleared up rapidly. On January 1st the left parotid gland became swollen, and the next day the right gland became involved. On January 5th, five days after the left gland became involved, she had some dizziness accompanied with nausea. On the 7th she had severe vertigo and vomiting.

She was seen by Dr. Wilson in consultation with her physician on the 8th of January, at which time she could not raise her head from the pillow without vomiting. The external canals and membrana tympani were normal. She was deaf in the left ear and complained of subjective noises; the noises still persist at times. The vomiting was not of the projectile type, and she had no fever at the time Dr. Wilson saw her. The vertigo lasted about two weeks, while the vomiting lasted only two days. At the time he saw her there was no nystagmus.

With the finger in the right ear she hears some sounds, but with the noise producer in the right ear she does not hear the

loudest shout. The static labyrinth is functionating, as shown by the caloric test.

DISCUSSION.

DR. DENCH said that these cases are not so very common, and he would like to know from Dr. Wilson what had been done for the patient therapeutically. Sometimes pilocarpin helps these conditions. He had seen certain cases of affection of the cochlea following mumps in which it had had a very beneficial effect. Just why the vestibular portion clears up and the cochlea does not, he did not know. The cochlear labyrinth has rather better circulation from the anatomic point of view than the vestibular. The only reason he could think of was that the internal structures are rather more delicate in the cochlea, and while the gross circulation of the cochlea is better, the anastomosis in the semicircular canals is more free. Perhaps another reason is that though we are apt to consider the three semicircular canals as being on three exact mathematical planes, they are not so, and each semicircular canal takes up a part of the function of the others. We may for this reason have a functionating static labyrinth while the auditory labyrinth is destroyed. He strongly advised using pilocarpin in this case.

DR. BLACKWELL, referring to independence of the cochlear and the vestibular branches, cited two cases which he had seen, in each of which the cochlea function was entirely lost without any history of vestibular irritation. Both presented perfectly normal vestibular action to both caloric and whirling tests.

DR. WILSON, closing the discussion, said that he had nothing to add excepting to say that he was familiar with Dr. Dundas Grant's paper on the use of pilocarpin, but understood that he uses it in the earlier stages of the condition.

Lymphangiosarcoma of the Concha.

DR. HUGH B. BLACKWELL: The patient, a boy, fourteen years of age, with a negative family history, was apparently in good health. The present trouble began eight months ago as a thin red line in the cochlea of the ear. This line gradually increased in size, raising and stretching the overlying integument, until nearly three months ago, when the skin covering became eroded and broke down; within a week the

tumor was converted into a raw bleeding mass. During the following two weeks the patient had three hemorrhages from the tumor, one of which was quite severe, lasting for an hour and only controlled by continuous pressure.

The patient first came under Dr. Blackwell's observation nine weeks ago in the otologic clinic of Dr. Robert Lewis at the New York Eye and Ear Infirmary. At that time the tumor was about five times its present size, filling the lower portion of the concha. Its upper half was devoid of any skin covering and bled freely when touched. At first the appearance was somewhat that of an ordinary aural polyp. There was no glandular involvement.

Closer inspection revealed the fact that the growth was attached to the floor of the concha and that the external auditory canal and membrana tympani were normal. The tumor was cocainized and removed as closely as possible to its base by means of a wire snare. Its removal was followed by a very free arterial hemorrhage which came from a large vessel located in the center of the pedicle. This was controlled by means of a pressure bandage, and the tumor was sent to the laboratory for examination.

Laboratory Report.—"I beg leave to report in the case of Thomas McH. (Clinic No. 3402) that the growth removed from the concha is covered with epithelium having a slight tendency to digitate. The main portion of this tumor is composed of irregular connective tissue cells in which is found a very liberal supply of blood vessels or channels, some with no walls whatever and the balance lined with a single layer of endothelium. Some lymphoid cells being also present, the histologic picture of lymphangiosarcoma is completed. I would add that pedunculated sarcomas in this region are not likely to recur if thoroughly removed.

(Signed) GEORGE S. DIXON, M. D."

Conclusion.—The condition is very rare. Politzer, in his book published in 1909, mentions only one case, that of Cohen Servaert. It is the first instance of its kind that I have observed in seven years' experience in two large active ear clinics. In connection with the previous history of hemorrhage, it is worthy of note that Jüngken states that in the simple angioma sometimes seen in this location a fatal issue may result from

the rupture of vessels. On admission to the clinic, the interesting significance of the case lay in its close resemblance to an ordinary aural polyp—illustrating as it does the necessity of a careful differential examination in these cases. Of course, it is recognized that the histologic examination of a tumor may lead to a widely different appearance from one based on its clinical picture. In view of Dr. Dixon's careful examination and the clinical evidence submitted, Dr. Blackwell believes that despite its present innocent appearance the growth should be regarded as an early malignant recurrence and should be extirpated by means of what now would constitute a comparatively simple operation.

Acute Mastoiditis—Perisinus Abscess, Three Months' Duration—Scant Middle Ear Symptoms.

Dr. Joseph Coleman: These two cases of mastoiditis which have come under my observation during the last few months belong to the class designated as atypical or insidious; atypical, in that the almost constantly existing symptoms which we are accustomed to find in acute mastoiditis, are either partially or totally absent; and insidious, because of the apparent slowness of the process, the coexisting extensive tissue destruction. I will report these two cases as briefly as possible.

Case 1.—Mr. P. M. B., aged forty-five years, came to my office December 22, 1915, complaining of inability to hear and pain on the left side, in the region of the mastoid antrum. From his history I learned that three months previously he had sudden severe pain in this ear, for which a paracentesis was done a few days later. Following the incision in the drum, he states that in spite of strict observation no discharge was seen. Examination of the hearing in the affected ear showed a watch which is normally heard at thirty inches to be heard at two inches, whispered voice at four feet, spoken voice at ten feet, Weber lateralizing to the left, the affected side, and Rinne diminished positive. The lowest fork heard was the 64, while all the high forks were heard. Upper tone limit normal. Examination of the ear showed the canal to be perfectly dry, drum intact, but slightly congested and thickened, resembling the condition found in a resolving acute catarrhal process. There was no bulging of the drum, and no sagging

of the bony canal wall. Patient presented himself three days later with slight swelling behind the ear, and diminution in pain. He was sent to the New York Eye and Ear Infirmary and operated the following day. No X-ray was taken.

There was found a good sized perforation of the cortex. The cavity of the mastoid was full of pus and granulation. The cellular structure was broken down through almost the entire extent of the bone. The inner plate was eroded, exposing an area of sinus about three-quarters of an inch in length, bathed in pus, and covered with a heavy layer of granulation. The cerebellum was also exposed, with quite a large exposure in the middle cranial fossa. Bacteriologic examination of the smear taken from the mastoid at the time of operation showed long chain streptococcus. Discharged from hospital January 4, 1916.

Case 2.—Mr. J. B. J. presented himself with a history of having had pain in the right ear five months previous. Paracentesis had been performed, but no subsequent discharge was noted by the patient. He has now a large swelling behind the right ear, which has existed for two days. The canal is dry, the drum membrane intact, pearly white, of normal contour, and there exists no sagging of the bony canal wall. There is no pain. Case seemed to be one of local infection. Patient was advised to enter the infirmary. At the time of operation a probe was found to extend deeply into the wound, and disclosed an area of bare bone. Suspecting a fistulous opening, the incision was extended, and an enormous perforation in the cortex was found over the antrum. The sinus had been exposed by the process from the knee to the bend below, where it turns in toward the bulb. There was also an exposure of the cerebellum on each side of the sinus. The entire area was covered with pus and dense granulation.

In this case there existed no indication of infection of the middle ear, though account was taken of the fact that a paracentesis had been done five months before. The cells of the mastoid were obliterated and the bone sclerosed. In the region of the zygoma the bone was soft and necrotic. There was no X-ray taken or blood examination made. Smear taken from the mastoid at the operation showed streptococcus capsulatus.

In both of these cases the process seemed to have undergone resolution in the tympanic cavity, then going inward and eroding the inner plate, coming to the surface later. In the second case, resolution in the middle ear had been so complete as to leave no indication of the infection. Undoubtedly the route of infection in both these cases was through the middle ear, yet in a case, particularly like the second, it can easily be seen that where the middle ear seems to take so small a part in the process, the route of infection might possibly have been considered to be of another source. Needless to say, primary infection of the mastoid must be exceedingly rare. Just why in some of these cases the process seems to pass through the middle ear and produce so few symptoms there is still a matter of speculation. It has been attributed to the anatomic structure of the middle ear or of the mastoid bone. Among conditions of the middle ear are mentioned thickened tympanic membrane, while as causative factors in the mastoid, the sclerotic type of bone, and bones with small aditus and far forward sinus are considered.

No doubt in many of these cases the inflammation of the tympanum may be so slight and transient as to escape notice, and passes on until it reaches the mastoid antrum, where inflammatory swelling in the aditus shuts off the tympanic cavity from the antrum, with proliferation of the infective material and destruction of tissue with consequent abscess of the mastoid.

The reasons for presenting these cases are the necessity of remembering the tendency of cases of this kind to run on until intracranial complications arise.

To illustrate the value of the X-ray, as no doubt in both these cases diagnosis could have been established much earlier by this method, and the necessity of bearing in mind these cases in which typical symptoms are absent.

DISCUSSION.

DR. MCCULLAGH said that he had had three cases within about a week corresponding very closely to those reported by Dr. Coleman. One case gave a history of having had an earache for an hour or so which cleared up and remained so for the six weeks before he came to the office. When first seen the

picture presented by the drum was one of a resolving catarrhal condition. The patient was watched for three or four weeks, without any change. He could hear a whisper at fourteen feet practically during all this time. Then one morning he telephoned, saying that he had had pain all night and that his ear was "sticking out from his head."

On examination a subperiosteal abscess was present, and on operation a large perisinus abscess was found, very much like that reported by Dr. Coleman.

A few days later another patient (a trained nurse) appeared with a subperiosteal abscess and practically the same history, except that the pain had been more marked. It cleared up under hot irrigations. The patient had had no symptoms for six weeks, when suddenly there was a rupture and perforation through the cortex. In this instance there was no erosion nor destruction of the internal table.

Dr. McCullagh said that at the same time he had another case under his care. This patient was a young man of about twenty years, who complained of a clicking noise in his ear, with a feeling of stuffiness. His drum was red, but there was no bulging nor sagging of the posterior superior wall. When he first saw this case the ear showed practically the same appearance of a resolving acute catarrhal process. The patient gave absolutely no history of any acute attack. He was watched for a month or six weeks, and then the other cases having come in in quick succession, there was some uncertainty as to what was going on in the mastoid. An X-ray picture was made, showing that the mastoid was filled with either pus or granulations. The condition was explained to the patient, and he agreed to have an operation as soon as he could arrange his business affairs, but he disappeared and had not been heard from since.

Dr. E. B. Dench said that everyone must recognize a type of latent mastoiditis in which an infection of some remote area of the mastoid occurs at the same time as the infection of the middle ear. Naturally, an acute otitis media is always an acute mastoiditis, but if—owing either to the topography of the mastoid in the individual case, or possibly to obstruction to drainage due to the inflammatory process—certain remote cells of the mastoid region are blocked off, the middle ear

inflammation may clear entirely, or almost entirely, and yet a deep seated focus remain in the mastoid. He had never seen a case in which the middle ear had cleared absolutely, although in most of these cases—socalled latent in character—the remaining middle ear involvement was so slight that one hesitated in opening the mastoid. In every one of these cases operated upon, rather extensive tissue destruction within the mastoid was found. The speaker emphasized the fact that an infection confined to the middle ear alone and those mastoid cells immediately adjacent to it, is limited to a rather small space. Such an inflammatory process, if so limited, will clear up in a comparatively short time if free tympanic drainage is instituted by free incision of the drum membrane. If within ten days or two weeks from the beginning of the attack the inflammatory process within the middle ear has not practically cleared up, it is then safer to enter the mastoid process. The speaker has followed this rule for the last two years, and has never opened a single mastoid without finding the destruction more extensive than he had anticipated. With a discharging ear, or a middle ear that has not returned practically to normal at the end of two weeks, the surgeon is safer inside the mastoid than outside it.

While radiography is of considerable value, it is impossible to tell from many X-ray pictures whether this cloudiness found in the mastoid is due to softening or to a beginning sclerosis. If after a number of months the cloudiness remains and the septa are intact, we know that the condition is one of sclerosis. If the septa are broken down, then we know that the radiographic changes are due to the presence of pus.

Dr. Friesner said that what Dr. Dench had stated was very true regarding those cases where there is middle ear discharge. However, there are cases which cannot truly be called latent mastoiditis so far as its being possible to suspect them by discharge. He then cited the case of a child, seven years of age, who had been brought into the Manhattan Eye, Ear and Throat Hospital with a fulminating acute meningitis. The child had never had any history of middle ear trouble until three days before Dr. Jones saw him, and all that he complained of at that time was a slight stuffiness in the ear. The hearing was good, the temperature was normal, and the appearance of the drum was normal. When seen with this fulminating meningitis the

light reflex and landmarks of the drum were normal. When the child developed a temperature, Dr. Jones did a myringotomy through what he believed at that time to be a normal drum There followed no discharge from the middle ear.

After admission to the hospital a lumbar puncture was performed. Pus and streptococci were found, and within a few hours the child died. A postmortem was done, which confirmed the antemortem diagnosis of diffuse purulent meningitis. Dr. Friesner said he chiseled through the antrum and found the tegmen soft, and the chisel dropped into a large cavity filled with mucopus. Then he shaved off the tegmen of the middle ear, and the ossicles were as clean as he had ever seen them postmortem.

Dr. Friesner said that he believes primary mastoiditis to be exceedingly rare, but this was an instance in which without any symptoms and little history of middle ear disturbance, or without anything leading one to suspect any complication, the child had an extensive destruction in the mastoid which led to the intracranial condition he had described.

He had also seen other cases. One of the surgeons at the Manhattan Eye, Ear and Throat Hospital had a trite saying: "Beware of the ear that is clearing up." Not long ago they had a case which looked like an acute catarrhal otitis, and was treated as such. Someone had done an ethmoid operation on the patient. While one of the men was examining the patient, he stopped breathing; he was operated upon, a subtemporal decompression first being done, and then the posterior fossa was examined. He had a cerebellar abscess which was opened. After the cerebellar abscess had been drained the mastoid was operated upon, streptococcus mucosus was found. The man's drum was faintly pink, no bulging; the landmarks were all present.

It is not the cases that have middle ear symptoms, etc., where one is so apt to go wrong; but where the middle ear symptoms are slight and where there is pain or headache and tenderness —that is where one must beware of ears that are clearing up.

A Fatal Case of Mastoiditis Following Pertussis and Influenza and Complicated by Meningitis and Concealed Measles.

DR. RALPH OPDYKE: Ordinarily mastoiditis follows one special infection, as influenza, measles, scarlet fever, pertussis,

etc., with, of course, staphylococcic or streptococcic organisms as the exciting factor; but seldom is a patient so unfortunate as to have so many of the exanthemata, as in this instance, precede the mastoidal involvement.

C. M., aged seven years, only child, and with healthy parentage. Mother living and well. Father died accidentally several years previously. Child's history practically normal since infancy till in the winter of 1914, when she had a severe attack of whooping cough which was of a protracted form and left her in a nervous and lowered resistant condition. Hardly had she completely recovered from this when she had a severe grippe infection which greatly aggravated her bronchial irritation from the earlier pertussis and attacked her gastric and intestinal tract in such manner as to make it difficult to feed her a sufficiently nourishing diet. This naturally added not a little to aggravate her abnormal nervous high tension and make her unusually sensitive to the slightest pain or noise.

As the grippe was subsiding her ears were attacked, first the right and then the left, and the frail little girl was in constant distress. She ran a rather unusually high pulse and temperature and seemed to have more than the ordinary severe earaches. Prompt incision of the drums relieved her, as is customary, and for a time it seemed as though her troubles were over. Though the left ear made a complete recovery, the right continued discharging, and the pain in the middle ear persisted and there was some tenderness over the right mastoid. As the blood count showed a relative high infection with continued lowered resistance and her general condition failed to respond, even though the aural discharge was mixed and only a very few streptococci present, an immediate mastoid operation was done on this right side. The findings were as follows: Cells but fairly broken down, only a few drops of pus present; the lateral sinus situated well forward and, when exposed from the knee nearly to the bulb, appeared normal in every way and was not interfered with.

Patient did fairly well, but the temperature, pulse and condition, both locally and generally, did not immediately improve as is usual. Though the wound remained perfectly clean and aural discharge scant, the temperature ranged from 103.5° F. to 105° F. at nights, and from 102° F. to 104° F. mornings, with a correspondingly high pulse. Respirations stayed about

thirty, and there was some cough, though the lungs were negative. Her throat kept very red and inflamed and bothered her. Cultures of same were negative for any specific germ. Measles was watched for, as she had been exposed to it a few days before the mastoid operation, but no rash nor coryza appeared, nor any other diagnostic symptoms. Another blood count was taken, showing leucocytes 15,000 and polynuclears ninety per cent. The various urinalyses were practically negative. Hiss serum was given at frequent intervals, also vaccines, but nothing reduced the pyrexia, nor improved her resistance. Some symptoms of an incipient meningitis were present, but all the various lumbar punctures were negative.

On the first of April, eight days after the mastoid operation, patient was removed home, as the advent of measles complicating was feared, the only symptoms of which was the suspicious-looking throat and the appearance of a rash after repeated hot baths. This rash disappeared and temperature again rose, and symptoms of a meningeal nature manifested themselves. Still nothing about the mastoid region gave any help, and the sinus continued to have a normal appearance, and there was no tenderness in the neck nor evidence of a thrombus being present. Several consultations by other aurists and pediatricians failed to clear up the picture to any extent, and the child gave further evidence of hidden measles with meningeal symptoms present. Her mind was perfectly clear and wonderfully alert up to within two days of her demise. Successive convulsions with cortical irritation now appeared, and a fairly typical erythema spread over her chest, face and neck. Both optic discs became injected and cloudy, and her vision failed to some extent. On the day of her death and some hours previous thereto she turned almost black with a typical advanced form of measles, and her eyes and nose gave every evidence of an advanced and tremendously severe form of infection.

During the last few hours she was in coma and her meningeal symptoms subsided entirely. No autopsy was permitted, and it is a moot question how much it would have helped to clear up the important factor as to whether the aural condition, existing for some time, had any active bearing in the later meningeal condition, or whether the latter was entirely

due to the exceedingly severe measles infection which did not declare itself, but on the contrary wellnigh burned up the little body, already weakened and with no resistance from the successive infections from within and without. Certainly, during the last thirty-six hours no one could possibly have mistaken the picture of a most virulent measles, but earlier the meningeal element was not in all respects plain—that is, insofar as resulting from a local and definite cause.

As an otologist I cannot believe resecting the jugular or excising the lateral sinus would have been of any avail. It is possible, of course, that the various cerebral veins were involved from the measles poison, but certainly none in such manner nor in a location whereby operative measures would have been of any avail. Also, that everything possible was done to hasten the measles' appearance and thus relieve the absorbing poison. The appearance of the mastoid when exposed, the child's continued cachexia, the later high blood count, and protracted varied illnesses, proved beyond peradventure the patient's tremendously lowered resistance, and account most conclusively how it was almost impossible for a favorable outcome to have ensued.

Bilateral Labyrinthitis With Brain Abscess Presenting Some Unusual Features.

Dr. S. McCullagh: Grace K., aged twenty-seven years, gave the following history: Fourteen years ago she had had a mastoid operation performed on one side, and one year later on the other side, for a chronic suppuration following scarlet fever. Has had a right facial paralysis since that time. There is still a discharge of foul pus from both ears, more profuse on the right side. Has been absolutely deaf since the operation. For some time past has been suffering from constant headache which varies in intensity. At irregular intervals, sometimes as often as twice a month, the headache will be very severe, this severe pain lasting from a day or two to a week.

Examination.—On both ears a radical operation has been performed. On the right side there is a postauricular fistula and the tympanic cavity is full of foul-smelling pus and granulation tissue. In the left ear there is also a small amount of pus and granulation tissue. There is complete loss of

function of both labyrinths. The station is very poor, even when the eyes are open, and the patient tends to fall to the right. A diagnosis of abscess in the region of the internal auditory meatus with chronic suppurative labyrinthitis was made and operation advised.

May 28, 1915, operation, assisted by Dr. A. Braun. Upon opening up the old wound and cleaning out the cavity, the external semicircular canal was found to have been eroded away. A Neuman operation was performed. The bone in the solid angle was softened and the whole labyrinth was full of granulation tissue. When the dura was first exposed there was no pulsation, but after the escape of some cerebrospinal fluid through the opening at the internal auditory meatus, pulsation was established. No abscess was found at this site. An abscess cavity, containing no pus, with firm walls, opening on the floor of the middle fossa just anterior to the knee of the sinus, where a mass of cholesteatoma had been lodged, was discovered. The opening was about a quarter of an inch in diameter and the cavity was about one inch in the vertical by one-half inch in the horizontal axis, as determined by careful probing. The opening was slightly enlarged and a drain inserted.

There was nothing of particular interest in the convalescence except the remarkably profuse discharge of cerebrospinal fluid, the abundance of which persisted for almost four weeks and did not cease entirely until about five weeks or more had elapsed. On July 4th she was discharged from the hospital and returned to her home, which is out of the city.

I saw her again on August 20th, when she gave a history of having had, for the past two weeks, "fainting spells," severe general headache, vomiting, petit mal, loss of memory, chills and a constant fever ranging from 99 2/5° to 101 2/5°. She was seen in consultation by Dr. J. A. Booth, who was of the opinion that she had a cerebellar abscess. Personally I thought that the sinus draining the old abscess cavity had closed before complete healing had occurred, or that there was a secondary abscess in that same region. The patient at first refused operation, but the pain became so severe that she consented to an exploratory operation.

On August 28th I reopened the old wound and removed a

portion of the squama. Great annoyance was caused by a profuse arterial hemorrhage from between the skull and dura that necessitated such firm packing for its control as to distort the relations. An incision was made in the dura over the lateral aspect of the temporosphenoidal lobe at the site of the former abscess, and with a long, narrow bladed knife seven exploratory punctures were made in various directions with negative results. The cerebellar dura internal to the sinus was then incised and five punctures made with a like result.

The symptoms were relieved by the operation and convalescence went along uninterruptedly until September 8th. The patient had been out of bed on September 7th. Early that morning the patient complained of violent headache. The pain rapidly increased in intensity and was accompanied by screaming paroxysms. She was very restless and vomited frequently. By noon she was unconscious and opisthotonos was well marked. The pulse was poor and respiration was of the Cheyne-Stokes type. The temperature was about 99°. Dr. Booth and Dr. Braun saw her in consultation with me, and we agreed that it was a case of fulminating meningitis. No lumbar puncture was made, as I felt it was unnecessary and uncalled for. A blood count showed leucocytes, 15,000; polynuclears, seventy-two per cent; large mononuclears, three per cent; small mononuclears, twenty-five per cent.

September 8th. Condition unchanged. Maximum temperature, 101°. Respiration, 14 to 30; pulse, 90 to 118. Blood count: Leucocytes, 28,260; polynuclears, ninety-two per cent; large mononuclears, six per cent; small mononuclears, two per cent.

September 9th. Patient conscious. Pain in head not severe. Vomiting persists, perhaps due to the morphin she had been receiving in large doses.

The nurse who had attended her after her first operation happened in and gave a history of a similar attack at that time, when the patient had been unconscious for four days with a well marked opisthotonos.

During further convalescence there were two similar but less marked attacks, accompanied by severe pain with screaming, vomiting and slight rise of temperature. The first attack lasted about five days, the second about two.

The patient was discharged from the hospital on October 4th.

I saw her last on February 4, 1916. The ear was completely healed. She still complains of a constant dull pain in the right temporal region, localized about one and one-half inches above the ear and over the right eye, in the region of the frontal sinus. She has attacks of what she describes as severe pain which come on at irregular intervals, have an indefinite duration—"usually all day"—and subside gradually. Otherwise she is well.

The only hypothesis that would seem to give a rational explanation of this history is that drainage from some undiscovered intradural focus of infection was temporarily shut off, precipitating the crises mentioned.

DISCUSSION.

DR. BLACKWELL asked if the abscess cavity had a wall.

DR. MCCULLAGH replied in the affirmative. It was evidently an old abscess with good rigid walls, which they were careful not to disturb.

Paper: Brain Abscess of Internal Capsule Following Injury to the Lateral Sinus During Mastoidectomy—Operation—Recovery.

BY WELLS P. EAGLETON, M. D.

DISCUSSION.

DR. DENCH expressed the opinion that so much had been said in the paper that there was little left to discuss. However, owing to the fact that Dr. Eagleton had been obliged to abridge his presentation of the case, one or two points were not quite clear to the speaker.

First.—He would like to know what the microorganism was which caused the trouble.

Second.—As he understood the history, the labyrinth was normal at the beginning of the attack, and thirty-six hours later was dead. (Dr. Eagleton stated that this was the fact.)

Dr. Dench was of the opinion that the serous labyrinthitis in this case was probably of mechanical origin, and was probably due to the sudden occlusion of the lateral sinus by a clot. This in turn might give a back pressure in the labyrinth and be

followed by an effusion of serum. Dr. Dench thought that as at the first operation the abscess was opened but not drained, the subsequent relief was very largely due to the decompression effect of the first operation. He could not quite understand how the patient escaped a purulent meningitis if the abscess was opened but not drained until six weeks later. At the second operation the drainage of the abscess through the center of the osteoplastic flap seemed to the speaker to be a procedure of distinct advantage. The speaker then detailed briefly a history of a metastatic abscess occurring in the island of Reil, in a case upon which he had operated and where the sinus had been opened at the time of the original operation. In this case the symptoms of involvement of the island of Reil came on about five weeks after the primary operation. The sudden occurrence of aphasia in the case, together with a high polymorphonuclear count, seemed to point to an abscess. Dr. Dench was urged, however, by the attending physician and by the consulting neurologist not to operate upon the patient, as these gentlemen looked upon the case as one of thrombosis. The operation was afterwards performed, too late, and although the abscess was drained the patient died. At the time of the operation the abscess proved to be an old one, as the walls were exceedingly firm. This patient gave a history of having had an attack of acute otitis ten years before. The speaker believed that the original purulent focus was deposited in the neighborhood of the island of Reil at the time of the first acute otitis, and that the second infection, ten years later, followed by the mastoid operation and the opening of the sinus, simply led to a lighting-up of the activity of this latent purulent focus in the speech area. The speaker's reason for believing this was the fact that well marked symptoms referable to an abnormal condition in the speech area developed every time the patient took an anesthetic; that is, the original attack ten years before the death of the patient occurred in Italy, and whenever the patient was given a general anesthetic she would immediately begin to sing songs in the Italian language, a language with which she was only slightly familiar and which she had practically not used since her visit to Italy, ten years before.

DR. SCRUTON said, in speaking of Dr. Eagleton's paper, Dr. Dench had said that the operation was practically a decompression, which Dr. Eagleton denied. He agreed with Dr. Eagleton in his position, for the reason that edema occurs whenever the brain is exposed, and if, as in this instance, the bone flap is replaced and the scalp sutured, it is not conceivable that much if any decompression is accomplished. The relief of symptoms in this case was entirely due to the evacuation of pus from an abscess cavity, and not to the lifting of the bone flap. The subsequent history bears this out as fact.

NEW YORK OTOLOGICAL SOCIETY.

Meeting of March, 1916.

Hemorrhagic Measles.

Dr. Philip D. Kerrison described a severe and unusual case of hemorrhagic measles which he had seen the same day in the Willard Parker Hospital. The patient, a girl of twelve years, came from a tenement apartment in which three other children of the same family had measles of the usual type. The patient under discussion was removed to the hospital on account of the unusual features and apparent virulence of the disease.

She had been ill three days, and there was a history of hemorrhage from the bowels (with the stools) and rather profuse hemorrhages from the nose, to control which both nostrils had been packed with sterile gauze. When admitted to the hospital she was also bleeding from the right ear. The ears had not previously discharged.

When seen by Dr. Kerrison the temperature was 103° and the patient looked profoundly ill. The skin from head to feet was covered with closely approximated reddish mahogany spots. In the mouth hemorrhagic spots were seen only on the roof. Both nostrils were packed with gauze. The right ear showed coagulated blood upon the drum membrane, which he did not remove. The left drum membrane presented an appearance which he had not seen in any other patient. It was intact, but showed the color changes due to blood filling the tympanic cavity. That is to say, the membrane, though reddened by contact with blood filling the tympanic cavity, did not appear to be inflamed, and the patient did not complain of ear pain.

Dr. Kerrison said that he had not incised either membrane, and had left instructions that the ears be left alone unless symptoms characteristic of tympanic inflammation developed. It was in his experience an unusual case, the outcome of which he would be able to report later.

DISCUSSION.

Dr. Harris said he saw bleeding from the ear a little over a year previously, occurring in an adult. The man had a history of repeated hemorrhages, before he was brought to his office, from the gums and from the nose. He was sent to the hospital and twenty-four hours after he had entered the patient had a very severe hemorrhage, and when Dr. Harris got to the hospital he found him exsanguinated and with hemorrhages in both ears. Examination of the drums showed, as far as he could make out, not trying to meddle too much with the condition, that both middle ears were filled with blood and the hearing very much reduced. The patient later had characteristic subcutaneous hemorrhages all over the body. The case was plainly one of purpura hemorrhagica. He tried to hunt for the source of the infection, and much to his surprise the bacteriologist succeeded in getting a pure culture of the Klebs-Loeffler bacillus from the tonsil. The conclusion Dr. Harris arrived at was that this case was one where the diphtheria was latent and yet where there was a sufficiently active organism in the tonsil to give rise to the blood changes and so to the hemorrhages. The process in the ear was allowed to take care of itself. The patient was given fifty to one hundred cubic centimeters of human blood serum, which promptly stopped the bleeding, but it must have taken two or three weeks for the blood in the ear to become entirely absorbed. Before he left the hospital the hearing was fully restored.

Dr. Dench asked whether this case did not resemble cases of hemorrhagic otitis complicating grippe. In grippe it was not uncommon to find hemorrhagic blebs in the canal, upon the surface of the drum membrane, and even hemorrhage into the middle ear. In some of the cases which went on to mastoid involvement, the mastoiditis was of the hemorrhagic type. Dr. Dench had always felt that it was the severity of the constitutional infection producing a toxin which was responsible for the hemorrhagic form of otitis in grippe. A similar condition might be found in diphtheria, scarlet fever, or any other severe constitutional disease.

Dr. Kerrison said that his case showed an absolutely different picture. It was hard to describe it, but the case had no

resemblance to the grippe ear. The blood was examined and there was not much change to be found.

Dr. Harris said that in his case there had been decided blood changes, with delayed coagulation and all the other evidences which Dr. Dench had pointed out.

Dr. Dench asked Dr. Kerrison if there had been any delayed coagulation in his case; if so, he would like to know what the time of coagulation had been.

Dr. Kerrison said that the delay in coagulation had been very slight.

Tubercular Mastoiditis.

Dr. Robert Lewis said he wished to relate the history of a case which was one of three similar cases, all of whom were born in the same institution within a few weeks of each other, in October. The three cases had been seen by different men, all had been operated upon and all had had a radical operation performed, which was in each case unfortunately followed by a facial paralysis. His case he saw in the latter part of December; the child, three months of age, had had a running ear for some few weeks before she was first seen by him. It was impossible because of the stenosis of the external auditory canal to see the drum membrane. There was a discharge coming from the ear of a thin bloody serum. He suggested operation at the time, but the mother objected seriously. He saw the patient at intervals of about two weeks for nearly six weeks or two months, and every suggestion of operation to the mother was met by refusal. Dr. Lewis advised her to obtain another opinion. She consulted Dr. Whiting some time later about her baby, and he advised immediate operation. A von Pirquet test, made by the family physician, gave a positive reaction. Dr. Lewis was then asked to operate upon the case.

He found the mastoid process filled with a mass of cheesy material, granulation tissue and necrotic bone; the posterior canal wall came away as a sequestrum. Very little curetting was done around the facial canal, but after the operation the child had a facial paralysis. It could close the eye, but in crying there was a distinct paralysis to be seen. The other two cases were also cases of tubercular mastoiditis. There was a cortical perforation and the periosteum covering the process was very thick, almost like cartilage. There was no evidence of a subperiosteal abscess.

Dr. Lewis said that this child had been partially fed by the bottle, and that the institution was very careful about the source of its milk.

DISCUSSION.

DR. DENCH said he would like to ask whether or not the facial paralysis had been present in the child before the operation was performed.

DR. LEWIS said he lifted the posterior canal wall right out, and this presumably exposed the nerve. From the fact that there was only slight paralysis of the upper portion of the face, he hoped it would eventually clear up. The child was three months old when he first saw it, and had been operated on only eight days previously. All of the middle ear was filled with necrotic bone and granulation tissue.

DR. BERENS said that sequestrums were very dangerous things in these cases, as one never knew how much of the nerve was exposed. He asked whether the evidence was that of a general tubercular infection or whether it was only local.

Mastoiditis—Vaccines.

DR. WILLIAM H. HASKIN said he recently had a very interesting experience in a child, where he had operated for mastoiditis in both ears, one year previously, which he believed had been reported before the society as a case of infection having come from an abscess in the jaw. The child became very well, both mastoids healed up beautifully, and the drum membranes were intact. In the latter part of December the child had an attack of grippe and an acute otitis media in the right ear. The drum membrane was incised and the patient got well. In the latter part of January the patient had an acute otitis in the left ear, but because of the mother having become ill the case was neglected for a week or ten days, when she showed a big posterior swelling. The drum membrane was bulging, but there was no breaking at all of the drum. Dr. Haskin made an incision through the membrane and behind the ear, but found the old mastoid wound filled with healthy looking bone, and he thought that pus had burrowed up along the superior wall of the canal. He did not do any further operation on the bone and watched her for a little while. A great amount of edema remained over the mastoid and cult-

ures were taken, a staphylococcus infection being found. The child received some injections of autogenous vaccine, and after the third injection the whole condition quieted down, the ear returning to its normal condition. Later the mother again brought the patient to Dr. Haskin, and this time the right ear was affected. The child was out playing in the snow and had gotten chilled, an acute infection having followed after two or three days. There was some swelling behind the auricle. The drum membrane was opened and the child immediately started on vaccines again. Much to Dr. Haskin's delight, after the third or fourth injection that ear completely recovered. He had seen the child at intervals of a week ever since, and there has been no further infection of any kind. The child had had ethmoiditis at the same time, which was treated with argyrol and suction. Dr. Haskin said he had been strongly tempted to go in behind that ear to operate, but did not think anything would have been found if it had been uncovered.

Dr. Berens said he would like to ask Dr. Haskin if there was no pus in the superficial tissue at all in this case, and if it was a socalled "white swelling."

Dr. Haskin said there was no pus, but a great inflammatory thickening. He said the swelling above the mastoid wound over the upper portion of the canal was a red and not a white swelling. He had fully expected to find pus, but did not.

Chronic and Recurrent Mastoiditis in Infants.

Dr. E. B. Dench said that within the last two years he had operated upon quite a large number of infants at St. Luke's Hospital suffering from recurrent or chronic mastoiditis. In some of these cases there was a history of intermittent discharge, while in others the persistence of the discharge was the cause for operative interference. In these cases the outcome had been eminently satisfactory, in marked contrast to the prognosis in mastoiditis in young infants given by Dr. L. Emmett Holt. In many of these young infants the operations were done not primarily for the relief of the condition within the mastoid, but simply because the children were in a marasmatic condition and no other cause could be found for the marasmus excepting the aural suppuration. In all of these cases, when the mastoid was opened, it was found to be the seat of a suppurative process, filled with granulation tissue and

softened bone. Immediately after the operation these children uniformly began to gain in weight. The same flattering results have followed the radical operation in young children where a profuse purulent discharge had persisted, and where the discharge itself, as well as the poor general condition of the child, was an indication for operation. In some of these cases, primary grafting had been followed by excellent results. In others, where the local condition was too foul for primary grafting, secondary grafting had been followed with benefit.

The speaker called attention to the fact that in many cases, especially in children, the middle ear lesion might clear up completely and a focus of infection remain in the mastoid antrum. This condition explained, undoubtedly, the many attacks of recurrent acute otitis with high temperature, so frequently seen in children. He also explained the cases of so-called fulminative mastoiditis sometimes observed—that is, cases with a history of acute otitis, with severe symptoms demanding mastoid operation within twenty-four hours after the middle ear became involved, and where upon opening the mastoid extensive destruction of the mastoid structures was found. The extensive destruction of the mastoid was undoubtedly not due to the immediate acute process, but was the result of several recurrent attacks of otitis, each attack causing a little more destruction within the mastoid until finally operative interference became necessary.

Dr. Berens said he would ask if any anatomic peculiarities had been found in any of these cases, and what the infecting organism shown had been.

Dr. Dench said that these cases usually showed a streptococcus to be the infecting organism.

Dr. Quinlan asked was not the infection through the pharynx; as many mastoid operations had been performed and the condition of the rhinopharynx had been overlooked and the culture field remained, it was likely that the infection could have easily been transmitted through the tube (if such was not obliterated at the time of the mastoid operation). Dr. Quinlan thought that the cleaning up of the pharynx had as much to do with success of the work as the major operation on the mastoid; even after the tonsils and adenoids had been removed, one must bear in mind that bad teeth had much to do with these infective conditions.

DR. DENCH said he wanted simply to call attention to these cases, as he had not seen anything written about them.

DR. HASKIN said the discussion bore out the same line of thought which was much in vogue about the focal infection of teeth. These patients with a focal infection invariably had a lowered vitality and marked anemia.

DR. BERENS said that at the Manhattan Eye, Ear and Throat Hospital the men operated on these recurrent cases in children without fear, and had not been scared particularly by bad prognosis. The results were excellent.

Osteomyelitis.

DR. WELLS P. EAGLETON said that some time over a year ago he presented a man, reported as a case of osteomyelitis, who was also presented before the Eastern Section of the American Medical Society. At that time he was asked by Dr. Dench if a Wassermann test had been made, and Dr. Dench told him that he thought the case was one with a syphilitic process from his experience of such cases. Dr. Eagleton told him he would have a Wassermann made. The man is now perfectly well, there was no return of the ear trouble, the incision had entirely healed and there was very little scarring. About one month previously Dr. Eagleton wanted to publish the case, but got hold of the patient's physician before he put the matter in print and asked this doctor to tell him how the patient was. The man was the father of three healthy children. The Wassermann had been three plus. Dr. Eagleton said he would not publish the case as one of osteomyelitis.

Cerebellar Lesions—Specimens.

DR. WELLS P. EAGLETON said that four months previously he had presented a specimen before the society, which he was now again showing as a sample of some of the middle ear symptoms that occurred in the pontal cerebellar angle tumors. During the last month there had come under his observation another such case, in which the otologic symptoms had also been thoroughly studied over a period of several months with the idea of a diagnosis. Dr. Eagelton said the one which the members had already seen emphasized the points of importance. The one cerebellar angular tumor was a dural endo-

thelioma. The man, prior to operation, had a marked nystagmus with the recognized tuning fork reactions. This case had been under observation altogether for a period of two years. There was very much doubt about the diagnosis of tumor in the second case, that of a child. Two very competent neurologists thought it was a case of hydrocephalus. At the autopsy a tremendous tumor was found. At no time did the child present any nystagmus, illustrating the point that they only had nystagmus in extracerebellar lesions and not in true intracerebellar lesions. Dr. Eagleton said that in a paper read before the American Triological Society three years previously, he called attention to the fact that in the vestibular reactions the opposite ear would be affected in cases of cerebellar growths. This child had been subjected to caloric reactions twelve times during a year and a half. At times there would be in the right ear an absolute absence of the reaction to the caloric. There would be no dizziness, no nystagmus, and the child would walk off without any symptoms at all. At other times, however, the reaction in one ear or the other would be very marked, and this would depend upon the condition of the child's head, as if the degree of the hydrocephalus had something to do with it.

During some days it would be suffering intensely. When the child was in good condition a reaction to the caloric could be produced from either side. Dr. Eagleton said this fact was interesting if it would tally up with some of the other otologic symptoms of the intracranial groups.

DISCUSSION.

Dr. Dench said he thought the loss of caloric reaction in these cases was due to increased pressure of the cerebrospinal fluid. The perilymph and endolymph communicate directly with the subdural space and with the ventricles of the brain. The speaker thought that in cases of hydrocephalus the ventricular fluid would be dammed back, and consequently, free circulation of the labyrinthine fluid would be prevented, thus accounting for the absence of the caloric reaction. He had seen two cases of cyst of the frontal lobe in which there was an absence of caloric reaction upon the same side. This absence of caloric reaction might be explained either by noncirculation of the cerebrospinal fluid or by an edema of the

vestibular nerve, due to pressure; in other words, we might have an edema of the auditory nerve or labyrinth, exactly the same as we have swelling of the optic nerve in cases of increased intracranial pressure due to tumor.

Regarding the remarks that tumors within the substance of the cerebellum did not cause nystagmus, he felt certain that he had seen cases of intracerebellar tumor in which nystagmus had been present. A recent author had stated that in cases of a tumor within the substance of the cerebellum, this symptom was not produced until the tumor grew to a sufficient size to become extracerebellar as well. Dr. Dench had no experience to confirm this statement.

DR. HASKIN said he would like to ask Dr. Eagleton what the nature of the tumor in his last case had been.

DR. EAGLETON said that the tumor was a glioma, although it had not been microscopically examined.

He said he thought otologists could be of assistance to the neurologic surgeons, as the caloric reaction, if applied properly, would help them add their mite of evidence toward the diagnosis of increased intracranial pressure, even without localized cerebellar symptoms.

DR. DENCH said that in certain cases decompression effects were certainly obtained simply by removing a portion of the calvarium without opening the dura. English observers denied that any decompression effect was possible without incision of the dura, and had performed certain experiments upon the dural sac (namely, attempting to dilate this with water) to demonstrate its absolute inelasticity. Notwithstanding these experiments, however, Dr. Dench was certain that in some of the cases where a large portion of the skull had been taken away and the dura had not been incised, a certain amount of decompression effect was obtained. He was very glad to agree with Dr. Eagleton in this regard.

Diphtheria—Hemorrhages—Mastoiditis.

DR. FREDERICK WHITING said he had recently had a case in a child, with an involvement of both ears. There was suppuration from both ears with a very large mass of glands under each ear and in the neck. The patient was running a very high temperature and was just such a case, Dr. Whiting

said, as he was sure most of the men present had at least once during the winter. There was a question as to whether the case with which he dealt was a simple middle ear condition or mastoiditis and sinus thrombosis. The temperature was of a fluctuating character and varied from 99 to 105 degrees, and the patient's appearance gave them great concern. The cultivation of the blood was negative, so Dr. Whiting concluded there was not a sinus phlebitis. After a reasonable length of time the physical appearances were such as to indicate the presence of mastoiditis, and a double mastoid operation was performed. The child did not seem to do well. The mass of glands in the neck appeared to diminish in no respect, and there appeared on the child's face and on the extremities quite a number of large ecchymotic spots. These spread and in the course of thirty-six hours assumed proportions which were anywhere from the size of a silver dollar or larger down to very small hemorrhages. The question arose as to whether or not the child might have scurvy, and in an examination of the mouth and gums Dr. Whiting discovered a small patch on one tonsil. He said that the throat had been examined day after day and found negative; but that morning in looking for hemorrhages on the gums, which he did not find, with the exception of one or two small ones, he saw the patch on the tonsil, which was gray and very suggestive. A gelatine, health-board culture tube trial was made, and by night there was a large gray patch on the other tonsil. When the report came back it was said to be the Klebs-Loeffler bacillus.

The hemorrhages in the meantime had extended pretty much over the body. No attempt was made to count them, but they appeared in great numbers, over the body, backs of the hands, arms, legs, face, palms, etc. The mastoids, as far as physical appearance went, looked very well; they were not granulating very freely, but there was no evidence to lead one to believe that such granulations as were forming were losing vitality at all. It was decided to do a transfusion in the case.

Dr. Lindeman of Bellevue did the transfusion, and a college classmate of the father of the child volunteered to supply the blood. This blood was replaced by a double amount taken from the donor.

The next morning it was with difficulty that one could recognize the presence or situation of any of the hemorrhages that

had been present the day before. In forty-eight hours there was not one to be seen, and in seventy-two hours one could not tell that the child had ever had any. The child's general condition improved in proportion to the skin condition; it went through a moderately quick recovery from the diphtheria, for which antitoxins had been administered, and although the adenitis persisted for three weeks or more, the patient finally made a perfect recovery and was discharged cured.

One of the features of the case, Dr. Whiting said, which was of rather remarkable coincidental interest, was the fact that the year previous this child's cousin, a boy of the same age, developed a double mastoid and diphtheria, and he had operated on him for double mastoiditis when the child had the tube in his throat. It was merely a coincidence, but seemed a most astonishing thing that the sons of two brothers, at intervals of exactly one year, should both have been subjected to a double mastoid and diphtheria, although the second one did not have a purpura.

DISCUSSION.

DR. DENCH said he would like to ask Dr. Whiting whether he thought the glandular enlargement entirely due to the diphtheritic infection.

DR. WHITING said that the glandular enlargement had been present fifteen days before the diphtheritic infection. The throat had been examined every single day because of the temperature, and never had presented any appearance which would suggest it microscopically. There was nothing in the throat, and of course as soon as he had seen the spot on the tonsil, a cultivation was made.

DR. DENCH said he was inclined to think a good many of these cases of enlarged glands coming on after middle ear infections were cases of sinus thrombosis where one failed to have bacteremia. Large glands over the bifurcation of the jugular vein, just at a point where the facial vein goes off from the internal jugular vein, it seemed to him, if there were a rise in temperature, might be a sign of a parietal clot. In a case he had seen, in which the child had not been operated on, although it actually got well, Dr. Dench thought there was a parietal clot in the sinus. He said a certain number of these cases of sinus thrombosis did get well without operation, but

there was no reason to suppose that a small parietal clot could not have existed in the sinus. Gland enlargement was really a life-saving measure, because it filtered out the germs from the blood.

DR. WHITING said during the winter he had had two cases of sinus thrombosis. They were both cases which came into the Mount Sinai Hospital at night, and presented the ordinary appearance of a subperiosteal abscess. Both of them were in children, one four years of age and one about three. Dr. Whiting said he was called and the cases were operated on before there was time to find out much of their previous history. They came in with a high temperature, a blood cultivation being made before the operation. In both of these cases, because of the very high temperature, Dr. Whiting opened the inner table and inspected the sinus, which appeared perfectly normal.

The next day, twenty-four hours later, both cultures were reported positive. The temperature in the meantime had dropped. As the children seemed to be in excellent condition, Dr. Whiting thought he would give them a chance and watch them. The next day the temperature did not rise, did not go above 102 degrees, so he thought he would wait another day, and so tided them along. Both cases made an uneventful recovery. Neither of these cases, bearing on what Dr. Dench suggested, had any large glands; certainly nothing which was enough to attract attention.

In view of these results, it would appear that the last word has not yet been said concerning the value of positive blood cultures in children.

DR. DENCH said he would like to ask if the tonsils had been previously removed in that case.

DR. HASKIN said that he had two patients at the Manhattan Eye, Ear and Throat Hospital, bearing out the same experience which Dr. Whiting had had, and which showed positive bacteremia, following mastoid operation, streptococcus hemolyticus, and he had asked Dr. Dwyer to operate on one of the patients. The patient developed a very severe erysipelas following operation. During the operation the sinus was not opened and the patient ran a temperature of about 105 degrees for several days. They had found two positive blood cultures, although positive blood cultures were not usually found in the

erysipelas condition. Dr. Haskin advised Dr. Dwyer to let the patient alone and the condition subsided, the erysipelas got well, and the patient was now alive without any trouble whatever.

Dr. Culbert had another patient which he operated on with the same history of a temperature of 105 degrees following operation. A blood culture was made, which was simply swarming with streptococcus hemolyticus. On the following day it subsided. A second culture was made four days later, but no bacteremia were found. He said he thought Dr. Berens had given orders that all cases of acute mastoiditis should have blood cultures taken, regardless of any indications for sinus thrombosis or not. That rule had been carried on for some time at Dr. Rae's clinic. He said he did not know the exact number of bacteremias, but knew that there were a number where the sinus had not been operated upon, but where the cases had recovered.

Dr. Eagleton said that Dr. Libman, who had contributed so largely to this subject, had told him that he had had one case where a blood culture was made, which after forty-eight hours was announced positive. The child recovered without further operative procedure. Dr. Eagleton said that a chill had occurred a number of times in such cases, and as far as he could make out, it uniformly occurred in children.

Dr. Duel said he would call attention to the fact that several years ago Dr. Jonathan Wright and he, at the Manhattan Eye, Ear and Throat Hospital, had made examinations for bacteremia, which they found present in a number of cases of acute otitis, chronic otitis and operated cases of mastoiditis not demanding further operation. He believed that bacteremia being present in many cases did not necessarily mean sinus thrombosis, nor did it demand jugular resection and sinus obliteration, in order to save the life of the patient. Often, on the other hand, after careful search, the bacteremia was never found, which it was thought must be present in suppurative conditions, the clinical manifestations of which so clearly indicated most radical surgical interference.

Dr. Berens said the bacteremias were not present in the demonstrable clot. This stand was the one that several others took, together with Drs. Jonathan Wright, Duel and Libman,

who had reported these investigations at the Otological Section of the Academy of Medicine.

DR. DUEL said he thought that Dr. Wright had the right opinion at the time of their investigation. He thought that the pouring out of the bacteria into the blood, producing bacteremia, was followed by a tendency of the blood to immediately demolish these bacteria. He believed it was not the tendency of the blood to form a culture medium for the reproduction of more and more bacteria, but that it was only from the local focus of infection that the bacteremia took place, and that one might frequently try a culture in a case where a bacteremia was actually present without demonstrating it. Only in certain very virulent types of infection would the blood not take care of the condition within a very short time. It, therefore, depended on the time a culture was made, the vein from which it was taken, and the amount of blood removed.

DR. WHITING said that Dr. Wright's proposition must contain a good deal of truth. He had operated on three cases during the past fifteen years, all of which were thirty years of age or over, and each one of whom gave a history of having had previous ear disease in childhood. When he operated he found an osteosclerosis, the densest portion being just over the situation of the antrum, where there had undoubtedly been a perforation of the cortex many years before, and where subsequently the wound had healed. There had been no operation of any kind, and the patients had come from that class of people who, if they were not operated in the hospital, had no other recourse for operation. The bone processes had healed. Each of these patients had come for operation many years after the earlier attack of mastoiditis.

Each of these cases had an epidural abscess, and when he cut away the skull sufficiently to expose the bed of granulating dura, he found the lateral and sigmoid sinus nothing but a firm, hard, fibrous cord.

The sinus had been obliterated for many years. Dr. Whiting considered that in the attacks in early childhood there had been spontaneous healing of the mastoid abscess through perforation of the integument, and that the sinus thrombosis had undergone spontaneous cure.

DR. DENCH said that undoubtedly certain cases of sinus thrombosis recovered spontaneously, with total obliteration of

the lateral sinus. He had seen at least one case of this kind in which no vestige of the sinus remained. Dr. Day, of Pittsburgh, had reported a series of these cases last year—either before the American Otological Society, or the American Laryngological, Rhinological and Otological Society.

Regarding Dr. Duel's statement, it was interesting to note that sometimes a blood culture from a vein in the arm would be negative, and yet if a culture were made from the infected lateral sinus this culture would be positive. (Dr. Dench had seen a case of this kind.) In other words, the blood possesses certain bactericidal properties, and although the blood contained microorganisms in the immediate vicinity of the local focus of infection, these organisms disappeared, owing to the bactericidal properties of the blood, in their passage through the general circulation. Dr. Dench pointed out the fact that it was not necessary to have a demonstrable clot in order to have bacteremia. In all cases of sinus thrombosis the infection begins in the outer wall of the sinus and then passes to the inner wall, a parietal clot forming at this point. A bacteremia would occur while the clot was parietal, but when the vessel became completely thrombosed the bacteremia would disappear—returning again only with the breaking down of the clot. It was then time to call in an undertaker, not a surgeon.

Dr. Duel said that, after all, we were coming back to the position of Dr. Gruening, who first called attention to these cases. He had said he operated on the clinical indications rather than on the laboratory reports. Frequently a case showed bacteremia which presented no alarming clinical manifestations and recovered without operation. Another case might be taking in a very small number of very virulent organisms and showing many urgent clinical symptoms which demanded obliteration of that portion of the vein near the infected area, whether a clot was preesnt or not. The arrest of the circulation in such a sinus was clearly indicated, to prevent general infection.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Regular Meeting, Held December 21, 1915.

DR. GEORGE W. BOOT, THE PRESIDENT, IN THE CHAIR.

Case of Atrophic Rhinitis Following Removal of Turbinates.

DR. GEORGE W. BOOT showed this patient for the purpose of getting an expression of opinion from those present regarding the class of work represented. The man was at some hospital in Chicago, where he paid a dollar and a half for a certificate, and he was to be taken care of if sick. He went to the institution with nothing very serious the matter, and they persuaded him to have an operation on his nose. His turbinates were removed, and they managed to extract fifteen dollars from him for the work. After leaving the hospital he was taken with a severe hemorrhage and brought to the County Hospital after he had lost a considerable amount of blood. At present he has a condition of operative atrophic rhinitis. He has a tinnitus in the right ear, which he did not have before operation. Dr. Boot's attention was attracted particularly to this case, because previously he had been very nearly involved as an expert witness in a malpractice suit in a similar case.

He wished the members would express their opinions as to what should be done in such a case; whether to keep still and let these men go on deforming noses in this way, or try to do something to have it stopped. In this case both lower turbinates had been very completely removed, especially the left side, followed by crusting.

DISCUSSION.

DR. JOSEPH C. BECK asked the speaker if he claimed an atrophic rhinitis in consequence of that operation.

DR. BOOT replied in the affirmative—an operative atrophic rhinitis.

DR. JOSEPH C. BECK thought an individual expression of

opinion might be given. His opinion is that atrophic rhinitis does not result from the removal of the inferior turbinates. In the days when the inferior turbinates were removed for all sorts of conditions, particularly ear conditions, he had seen something like forty cases while visiting in Louisville, in Dr. Dudley Reynolds' practice, in which he (Dr. Reynolds) had removed lower turbinates on both sides. These cases were all the way from one to three years after operation, and in none of them was there any crusting in the nose, or anything that looked like atrophic rhinitis, that we are accustomed to speak of as such. They all had more or less pharyngitis sicca, with the deep injected and glazed smooth posterior wall of the pharynx. They did complain of lack of air rather than too much air, but he thought all operators had had that experience with these cases. As to removal of the lower turbinates as a causative factor of atrophic rhinitis, he would take issue with Dr. Boot.

Dr. Alfred Lewy did not see the case, but from a medico-legal standpoint, he would like to refer to a case he saw in which both lower turbinates had been removed by one of the best known general surgeons in Chicago. In this case the patient was troubled with persistent crusting for many years after the operation.

Dr. A. H. Andrews thought it would be interesting to know the condition of the nose before the operation in Dr. Boot's case. If there was atrophic rhinitis soon after the removal of the inferior turbinates, he would be inclined to think that the same process existed before they were removed. He does not believe in the indiscriminate removal of the inferior turbinates. Such practice should be severely condemned. With Dr. Beck, he questioned whether real atrophic rhinitis is ever caused by the increased lumen of the nostril. It requires other etiologic factors to produce the pathology.

Dr. J. Holinger thought that a Wassermann and a systematic washing of the cavities would be of importance in such a case before making the diagnosis of atrophic rhinitis. There are scars in the nose that seemed to him to have the typical star shape so characteristic for scars after syphilitic lesions.

Atrophic rhinitis is essentially different from a specific

rhinitis, or rhinitis due to suppuration from different cavities. These diseases ought to be excluded before a diagnosis is made, since either can cause crusting.

DR. BOOT, in closing the discussion, said he did not mean to give the impression that he thought the pathologic changes of atrophic rhinitis were present in this case. It is a case, however, where there is a very marked condition in the nose, as a result of the operation, and he has some of the clinical signs of atrophic rhinitis. He did not mean to say that it was an atrophic rhinitis from a pathologic standpoint.

In his opinion, the case certainly does not resemble a case of syphilitic ozena.

DR. BOOT, in the absence of the other two cases which he had expected to demonstrate, exhibited a specimen which he thought would be of interest. It was removed postmortem from a man of about thirty-five years of age, who had been in the County Hospital off and on for a year or two. He entered first with the diagnosis of syphilis of the larynx, and was under Dr. Friedberg's care. He recovered sufficiently to leave the hospital, but returned after a year or so. At this time he was very dyspneic, and it was necessary to do an emergency tracheotomy. It was never possible to dispense with the tracheotomy tube. After wearing it for about a year he came back again, desiring to have somthing done so that he could breathe through the larynx. He could not get any air into the lungs. He could exhale and phonate, but no air would go in. On examination, the speaker could not find an opening through the larynx, and was unable to pass any sound through, so he went in in the other direction—through the old tracheotomy wound—and as he supposed cut into the larynx, but cut into this cavity (illustrating on specimen). Very shortly after this, before he had succeeded in dilating the stricture, the man began to cough out any substance he would drink through the tracheotomy tube, and he was then turned over to the general surgeons for a gastrostomy, so that he could be nourished. He did not rally from the operation of gastrostomy, but died on the third day, and at the postmortem the specimen was removed. The speaker passed a toothpick through the opening from what he supposed was the lumen of the larynx at the time of the laryngofissure, but which may or may not have been that. At the postmortem the posterior part of the larynx

was found in this position, and an opening below was found where the tracheotomy tube had ulcerated through. He has been unable to find the arytenoid cartilages in the specimen, and not much of the thyroid cartilage. An interesting feature of the case was that at the postmortem he cut into the thyroid gland and found a round, whitish area, which he supposed was a gumma. A piece was cut out for sectioning, and afterwards reported to be a carcinoma.

Carcinoma of the Mandible—Exhibition of Specimen.

DR. L. W. DEAN, Iowa City, Iowa, exhibited a specimen of squamous celled carcinoma of the mandible, without any ulceration of the mucous membrane of the tongue, floor of the mouth or lips. This condition was operated on by the method devised by Dr. Prentiss for the removal of half the mandible without interfering with any of the branches of the facial or producing any deformity about the mouth. An interesting feature in connection with the case is the appearance of the patient at the present time, shown in the photographs passed around.

This patient came with a history of having had, in October, 1914, a small, warty excrescence removed from the angle of his mouth. Following this operation the wound healed quickly. The speaker was unable to get the excrescence which was removed for microscopic examination. Naturally, he assumed that it was this squamous celled carcinoma. One month after its removal the patient had a tender premolar. The excrescence was on the right side and the tender premolar was in the mandible of the right side. Almost all patients with malignant growths in the mandible, and many of those with malignant growths in the superior maxilla, give a history of having had teeth extracted for pain, and the dentist telling them that the tooth was normal. This patient had this premolar extracted a month after the operation on the excrescence, and the dentist said it was a vital tooth, so the speaker assumed that this malignant growth was developing at that time in the center of the mandible. Following the removal of the tooth the patient had no trouble until last June, when he felt a little nodule on the border of the ramus, below the premolar on the right side.

When the patient came under Dr. Dean's observation he had a growth of some size—probably the size of half a hen's egg—which was not tender on pressure. It was very hard. Wassermann examination negative. X-ray examination showed absorption of bone. There was no ulceration at this time of any of the mucous membranes of the mouth. Since seeing this case, Dr. Dean has gone over the literature on the subject very carefully, and has found the statement frequently made that whenever you have a squamous celled carcinoma of the mandible, you always have an ulceration of the mucous membrane, either of the tongue, floor of the mouth, alveolar process or lip. The fact that there was no ulceration of any of these membranes at the time would suggest that it was not carcinoma. A piece of the growth was removed, however, after permission for operation was secured, providing it was found to be malignant, and examination showed it to be a typical squamous celled carcinoma.

In the one patient from whom Dr. Dean had previously removed half the mandible there was the usual postoperative drooping of the corner of the mouth and some flow of saliva over the chin and noticeable deformity. He consulted Dr. Prentiss regarding the operation. Dr. Prentiss advised the following operation, which was performed: Incision in the median line, beginning at the lip, going down over the chin, and then lower down on the neck, extending backward and upward, and terminating at least three inches below the ear. This incision extended through the skin, superficial fascia and platysma. These structures were lifted up to the inferior margin of the ramus. Then a second incision was made deeper, and under the submaxillary gland, the gland lifted up until the inferior margin of the ramus could be felt under it. Then the operator had in one mass the submaxillary gland and the lymph glands in the neighborhood. The outer flap was composed of skin, the superficial fascia and platysma. Then the muscle attached to the inner surface of the mandible was severed, and the flap from the margin of the mandible dissected upward, cutting it as close to the mandible as possible without cutting any of the infected tissue, lifting up the mandible so as to carry in the flap every branch of the facial nerve. Using a ribbon retractor, it was quite easy to bring the whole mass

up, so as to expose the temporomandibular articulation and disarticulate the mandible. Drainage tubes were placed below, so that the maximum scar is at the point fairly well concealed when the wound has healed.

The impression seems to prevail that these patients after removal of half a mandible are in a very serious condition. This man, as a matter of fact, says he is enjoying himself just as much as ever. If he were to enter a room nobody would notice that the operation had been performed.

After this operation the speaker had the opportunity of having Dr. Wilson, of Cleveland, see the case, and he expressed the opinion that it was not advisable to attempt to make anything to replace the half jaw.

The prognosis in this case Dr. Dean considers to be good. Before the operation he had felt the chances were good, and had urged the man to have it done because of the fact that it had been slow growing, and with the exception of the glands right between the submaxillary gland and the ramus there was no involvement of the lymphatics, so far as he could determine. At operation, search was made for enlarged glands in the neck, and none were discovered.

Specimen of Giant Celled Sarcoma of Jaw.

The second specimen was exhibited by Dr. Dean because of the first one. It was a giant celled sarcoma of the jaw, section of which showed how the growth did not involve the bone. The speaker's conception of tumors of the mandible is that the socalled giant celled sarcoma is the only sarcoma where we are justified in not removing one-half of the mandible. The only other malignant tumor where we are justified in not removing one-half is an epithelioma involving the alveolar process.

DISCUSSION.

DR. GEORGE W. BOOT asked Dr. Dean what he did to control hemorrhage in the first case reported.

DR. DEAN said there was practically no hemorrhage, and what hemorrhage there was could be controlled easily by the use of the hemostat.

DR. JOSEPH C. BECK asked if there was any X-ray picture taken.

DR. DEAN replied that there was, and it was very suggestive of a cyst of the mandible.

DR. BECK asked if the X-rays had been used therapeutically, to which Dr. Dean replied in the negative. The only use of the X-rays had been to take a picture before operation.

Paper: Pathologic Affections of the Cavernous Sinuses.*

BY H. G. LANGWORTHY, M. D.

DISCUSSION.

DR. L. W. DEAN, Iowa City, Iowa, said that in connection with disease of the cavernous sinuses he had had three experiences. The first case was one that came into the University Hospital about three years ago with a septic temperature. There was no exophthalmos. There was optic neuritis on the right side, but the patient had a very bad chronic suppuration of the sphenoid. That was all that was found. Within two days, however, he had a marked exophthalmos, and died about four days after first coming under the speaker's observation. At the time he was first seen he was a walking case with a septic temperature, chills and fever up to 105 degrees, then dropping to normal. Postmortem showed just as typical phlebitis of the cavernous sinus as one ever saw in the lateral sinus, with suppuration of the mastoid. There was a necrotic area of the wall, a dehiscence of the inner wall of the sphenoid, and over this dehiscence there was a necrotic area of the cavernous sinus, with the septic clot. Postmortem made the diagnosis positive.

That case has been of interest to Dr. Dean because there was positively no trace of exophthalmos or edema of the nerve head when the patient was first seen.

The second case was one of arteriovenous aneurism that had been seen by Dr. de Schweinitz, and the diagnosis confirmed by him. Two years ago the speaker ligated the common carotid in that patient, with a fine result. This was a traumatic case, with the pulsating exophthalmos accompanied by terrific pain. By "fine result" he meant that it was fine so far as the pain and slight diminution in size of the tumor were

*See page 560.

concerned, comparatively speaking, the principal factors in a "fine result" in a case of this kind. This patient is of especial interest to Dr. Dean now because he has a beautiful collateral circulation.

About two months ago he went through the literature to find out just how this collateral circulation was established, and talked with Dr. de Schweinitz, who everyone knows has done more work than anyone else in the world on pulsating exophthalmos, and his opinion regarding collateral circulation was not very definite. Dr. Dean had Dr. Prentiss look this second patient over about a month ago, and the conclusion was that the collateral circulation was mostly through the vertebrals. As a matter of fact, he said, it was not a definite opinion, but that was the best he could give.

The third case was one that was reported before the society, and occurred following tonsillectomy many years ago. The patient had a septic temperature coming on after exposure. He ran away from the hospital, exposed himself, and got an infection. When seen by the speaker, his temperature was 105 degrees. He had exophthalmos on the right side. The temperature was typically septic. Optic neuritis was also present on the left side. This patient recovered without a doctor, and came to Chicago afterwards and had the right eye enucleated. Dr. Dean has always thought the case was one of cavernous sinus phlebitis which recovered, but has had no definite way of knowing whether it was that or a sepsis of the orbit.

Dr. Joseph C. Beck said it had been a pleasure to listen to Dr. Langworthy's paper, in contrast to the paper of a similar topic which he read before the Ophthalmological Society, which consisted of a large number of data, and which was absolutely of very little value to the speaker. The paper presented this evening was entirely different. It was practical, and while on a rare subject, yet he was sure that all the members appreciated the work done by the essayist.

The question of intermittent exophthalmos, while perhaps not the subject for discussion, was interesting. Very recently Dr. Beck has had a case of intermittent exophthalmos in which he made an exploratory operation, or rather, was preparing to do an exploratory operation on the orbit for a possible tumor

within the orbit. There was, however, nothing definite to show this condition. This patient had been studied carefully by ophthalmologists, with no evidence of any intraocular disease. The condition had no effect whatever on the vision, but it was stated by the physician in charge that every time the patient became excited, nervous and tired, she got an exophthalmos. Dr. Beck never saw it himself, but when the patient was put under the anesthetic, and while he was preparing for operation, he looked around to see the face of the patient, he and all present saw the disproportion of the two eyes. He scrubbed up as quickly as he could and examined the eye; he felt above the inner canthus, and deep in the orbit he felt a mass something like a rubber ball. He, therefore, decided that there probably was a tumor—maybe an angioma—within the orbit, and on the nasal side. Nasal examination was always negative. X-ray examination did not give any hint as to the condition. Incision was made, as would probably be done for a Killian, only further inwards in the orbit. As soon as he came on to this mass, a slight opening was made, and a real gush of pus went all over the face of the patient. This pus was reddish yellow in appearance, like innocuous pus. After wiping out a quantity of this material and exploring further, he found that the entire ethmoid had been destroyed. The orbital periosteum was intact—no erosion—but thickened. In other words, it was a case of infected cyst of the ethmoid, both anterior and posterior, reaching clear back. Outside of the intermittent exophthalmos there was nothing unusual in this case, and he wished to call attention to the fact that one might have suspected in such a case a cavernous sinus condition, as mentioned by the essayist. If he had heard the paper of Dr. Langworthy before he operated, he would have thought his patient had perhaps such a condition of the cavernous sinus. The bacteriologic examination of the pus was pneumococcus, and the subsequent course was that after a middle turbinectomy and ethmoid opening. The patient made an uneventful recovery.

Dr. Langworthy referred to arterial and venous conditions where the carotid artery and cavernous sinus were exposed in the sphenoid sinus. He (Dr. Beck) once had a specimen from Professor Hoove, of Iowa, in which the carotid artery and a portion of the cavernous sinus were free in the lateral wall of this sphenoid sinus.

In regard to the point made of injuring the cavernous sinus in a Gasserian ganglion operation, the speaker had operated one such case, and in severing the posterior root he could very clearly see the cavernous sinus and see how easily one might injure it in that operation.

Dr. Dean, in his discussion, mentioned the ligation of the internal or common carotid artery. Only three days ago the speaker operated on a patient for cancer and resection of the lower jaw and part of the soft palate. He was forced to ligate the common carotid artery. There followed a hemiplegia, with the patient in good condition after operation, and within twelve hours after operation the patient died, apparently just because of his heart giving way. While he recovered consciousness, he could not speak after the operation.

Two months ago he did a similar operation on the lower jaw and part of the upper in a malignant case, doing a temporary compression on the common carotid artery, but it was not correctly done. The forceps were applied too firmly, and on relieving the artery forcep he noticed a distinct compression of the artery, which he feared would necrose, and possibly a fatal secondary hemorrhage ensue. He, therefore, ligated the common carotid, and that patient had the same symptoms—hemiplegia—with death following.

Looking up the literature, he found that twenty-five per cent of cases of ligation of either the internal or common carotid die from just such symptoms, and he wished to say, while this was not a point in discussion, that he would keep away from such procedure in the future.

He wished to call attention to the reaching of the cavernous sinus very easily by a procedure first proposed by him for reaching the hypophysis, namely, through the antrum, being able to reach the opposite side. For instance, in operating on the right cavernous sinus he would go by way of the left antrum of Highmore, in through the sphenoid, and then on to the cavernous sinus on the opposite side. It would certainly be an easy method by which we could reach it. He has done it on a cadaver a number of times, and in two cases on the living, in operating on the hypophysis.

DR. HARRY KAHN referred to a case which a general practitioner operated some few years ago for the removal of tonsils and adenoids, but did not get the adenoids out completely. Dr. Kahn operated on the adenoids under a general anesthetic, and sent the patient to bed. About twelve hours after operation the right eye began to get larger, and finally there was a proptosis. The eye was greatly swollen and looked like a plum. The patient had a septic temperature for two or three weeks, and then, by expectant treatment, the whole thing disappeared. The speaker did not know whether that was a cavernous sinus affair or not. He was unable to make a definite diagnosis. Dr. Frank saw the case with him. There was no pus in the nose, and no evidence of anything except the proptosis, septic temperature, and meningeal symptoms evidenced by headache and vomiting. Treatment consisted of urotropin, hot applications, and so on, and the patient entirely recovered without any loss of sight or disturbance of any sort.

He felt it was worth while to report such a case following an adenoid operation.

DR. J. HOLINGER was very much surprised to hear nothing in the paper, nor in the discussion, about the progression of the thrombosis to the cavernous sinus from the lateral sinus in mastoiditis and inflammation of the ear. We know that the progression of the thrombosis to the cavernous sinus and other sinuses of the base of the brain is one of the most frequent causes of death in cases of thrombophlebitis of the lateral sinus. Certainly he would think that this would be at least as important as the eye symptoms, exophthalmos, and so forth. The progression of the thrombophlebitis in that direction is discussed to a great extent by any number of ear men, and especially Leutert, many years ago, in his strong plea against ligation of the jugular vein in cases of thrombosis of the lateral sinus. He made the statement that undoubtedly most of the cases of progression of thrombophlebitis along the cavernous sinus from the lateral sinus might be avoided if (1) the lateral sinus were not handled so much in the operation, and (2) if the jugular vein were not ligated. He advised simply to lay bare the lateral sinus to as large an extent as possible, but otherwise leave it alone. Not even the splitting of the sinus is advocated.

This is the process which brings ear surgeons most frequently in contact with diseases of the cavernous sinus, and we have learned to look at any reliable sign of thrombosis of the cavernous sinus as a pretty sure signal of a fatal termination of a case of sinus thrombosis.

DR. GEORGE W. BOOT had had one case of thrombosis of the cavernous sinus in a young civil engineer, who was working out in stormy weather. He had a furuncle on the side of his nose and was brought to St. Francis' Hospital in Evanston with a well-marked thrombosis of the left side, which very shortly—in the course of twenty-four hours—extended to the right side, so that the eyeball protruded a great deal, with a great deal of ecchymosis. This man had meningeal symptoms, and in two or three days died of meningitis.

Shortly after this he saw a boy of about eleven years who had an exophthalmos of the left eye. He had a chronic suppurative otitis media on the left side. In this case those who saw the patient thought he had the condition referred to by Dr. Holinger, namely, an extension of the thrombosis from the lateral sinus to the cavernous sinus. Accordingly, a radical mastoid operation was performed, but failed to relieve the condition. Shortly after it became evident that the trouble was not in the sinus, but was a sarcoma within the orbit.

Dr. Boot saw a third case not long ago, in which the question of thrombosis of the cavernous sinus was considered, but finally ruled out. The final diagnosis was that of inflammation of Tenon's capsule, although there was no tenderness of the globe on pressure.

DR. LANGWORTHY, in closing the discussion, said there was not a great deal to be added to what he had already said in his paper. In a single evening it was quite impossible to cover all of the points which might come up in a discussion of the cavernous sinus, and especially its relation to other parts of the head. While he had been interested in this subject for a long time, it is very difficult and almost impossible to get material to work with, and our knowledge of conditions must be accumulated in the way brought out in the discussion. The object of his paper was to systematize the cavernous sinus and treat it as a separate organ, if possible, and to classify the pathologic effects, which task had not been easy. He realized that it was not a practical field for labor, but with the attention

that the pituitary body is receiving, and will continue to receive, it seemed to him that that would probably throw more light on the cavernous sinus and the structures adjacent to the sphenoid bone.

Stereoscopic X-ray plates, for instance, ought to be of help in clearing up a great many possible affections of this sinus. He has seen some of the most beautiful plates in Dr. Beck's clinic, and they have certainly been most instructive, and have suggested the possibility of obtaining definite knowledge beforehand in certain affections, and when one stereoscopically can look into a head, as it were, and see the lines of the bones, just as though it were a glass bowl, it is certainly a wonderful sight. There is probably no limit to the heights that can be reached along these lines.

Dr. Dean's experience with several cases in some ways is unique. The speaker doubted whether many present had seen many cases of thrombosis. Some of Dr. Dean's reports had been incorporated in the tables which accompanied the paper.

Regarding Dr. Beck's remark, especially relating to the paper read before the Ophthalmological Society, that was a paper which dealt purely with thrombosis and its relation to the future; that is, the matter of possible early diagnosis, and the various ways of reaching the cavernous sinus, all of which, however, have still to be worked out. Dr. Beck's method is certainly as practical as any. Dr. Langworthy had attempted to work this out himself, and it certainly seems that some method of going upward through the nose might be one of the first things thought of in dealing with thrombosis.

Regarding Dr. Kahn's interesting case, he hardly felt like commenting on it, from the meager reports which he gave. It was evidently one in which a diagnosis would be very difficult, and he would want to have more complete information.

With reference to Dr. Holinger's remarks, he wished again to say that the paper had to deal with the matter of classification, and was not an attempt to cover the single subject of thrombosis, which would require a good many pages.

Whether anything practical would come out of the study of the cavernous sinus or not, he did not know, but it seems to him that in dealing with some of the cases, a few of them would come under the surgeon's care, and possibly be relieved by operation.

Paper: Two Severe Cases of Ulceromembranous Angina.*

By Ira Frank, M. D.

DISCUSSION.

Dr. Robert Sonnenschein thought the paper of Dr. Frank very interesting and valuable for many reasons, but particularly in pointing out two important facts. First, the marked clinical contrast between cases of socalled Vincent's angina, and, second, the fact that fatalities may occur in this disease. To judge from the literature, one would imagine that these cases are practically all favorable in their prognosis. It was his privilege to see at least once both of the cases mentioned in the paper. The first one was already in the stage of healing when he saw it, but the second case was seen one day when Dr. Frank was out of the city, at which time an attempt was made to introduce food into the stomach, owing to the fact that the patient was unable to swallow in the ordinary way, by passing a very fine tube-like catheter through the pharynx. This failed, however, owing to the fact that the patient immediately began to complain of severe pain.

Last spring Dr. Sonnenschein saw two cases in the Skin and Venereal Ward at the Cook County Hospital, both men, one white and one colored, and both died. This disease may and oftentimes does end in death.

Just one point further that is not entirely relevant to the subject, namely, the name of this disease—Vincent's angina. It seems to him that in justice to a man who has done the work the name ought to be changed. Arthur Meyer wrote a large monograph on this subject, and showed that it was Plaut who described the organisms a year and a half before Vincent. Owing to the fact that the latter got his name into the literature, the disease has been called after him.

Dr. Alfred Lewy reported, from a very limited experience with the milder form of ulceromembranous or Vincent's angina, that sodium perborate has been very efficacious in its treatment. He has given up the trichloracetic acid application, simply cleansing the ulcer with peroxid and swabbing, and using a perborate gargle, and application of perborate of

*See page 637.

sodium powder, with great relief, so far as discomfort was concerned, and great help in rapid healing.

Dr. George W. Boot said that the two cases referred to by Dr. Sonnenschein at the County Hospital he had had the pleasure of seeing twice. A third case he had expected to show to the society this evening—a colored woman of about sixty years, who entered the County Hospital with very marked ulceration, with formation of Vincent's membrane over the posterior brim of the pharynx, tonsil and pillars of the pharynx. Smears gave the spirillum and fusiform bacillus. The patient failed to improve under local antiseptic measures. Wassermann examination positive. She was put on antisyphilitic treatment. The ulceration healed, but with a stenosis of the nasopharynx. There was only an opening of perhaps one-half centimeter in diameter between the oropharynx and nasopharynx. He is at present attempting to restore the opening, following the method of Rose, of putting a stitch through this small opening remaining and bringing it out at the side, about where the normal location of the side of the pharynx should be, tying the silk stitch and leaving it in for a considerable time, hoping the epithelium will line the canal. Then he expects to divide the tissue.

Vincent's angina, socalled, it seems to him is hardly a distinct entity, in that the organisms of Vincent's angina are present in so many different conditions in the mouth, without making a distinct disease. They are found in carious teeth, and he has seen smears from diphtheria patients, from the false membrane, give large numbers of the spirillum and bacillus, which were mistaken for the fusiform. So that he thinks we can consider Vincent's angina as primarily a deposit on another process; that is, in any ulcerative condition in the mouth you can get the same condition as in Vincent's angina, without it being a distinct disease.

Dr. Joseph C. Beck said that Dr. Boot's discussion reminded him of a case he saw in consultation, in which there was a fatal result in an ulceromembranous angina. When he saw the case the history was given that the specific treatment was applied, namely, painting with ten per cent carbolic acid solution, daily, probably for a week or so. He could readily see where the use of that remedy might enhance the action of the

infective organism by lessening the vitality of the tissues and producing a local necrosis, and then subsequently this ulceration. He would like to know whether there was anything like that used in the cases reported.

Second, he wished to call attention to the work of Professor Siebenmann on ulcerative sore throat from the leptothrix. Four cases of that same disease, ending fatally, were reported by Brown Kelly, of Glasgow. Was anythink like that looked for in the cases of Dr. Frank? Did Dr. Frank make any report on the bacteriologic or smear examination?

This condition described by Dr. Frank is known as Plaut-Vincent's angina, and it is also known that we find the leptothrix in the mouth, especially when it is in an unhygienic condition; in cases of low vitality of tissues, and general debility, the leptothrix will thrive to the extent of producing marked ulceration. It is well worth while to bear that in mind in treating Vincent's angina.

DR. FRANK, in closing the discussion, said, in answer to Dr. Beck, that a smear and culture were taken, but nothing particular was found.

Answering Dr. Lewy, he had never tried the remedy referred to.

Salvarsan has been used in cases of this character, both locally and subcutaneously, but he did not use it, for the reason that it is too expensive now.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Regular Meeting, Held February 29, 1916.

DR. OTIS H. MACLAY, THE PRESIDENT, IN THE CHAIR.

Histologic Specimen of Tumor Removed From Larynx, Together With Remarks on Direct and Indirect Laryngoscopy.

DR. NORVAL H. PIERCE reported the case of a man who came to the Illinois Eye an Ear Infirmary complaining of hoarseness. There was no pain. Dr. Schoolman made a diagnosis of tumor of the larynx, situated on the left vocal cord, very close to the anterior commissure. Direct laryngoscopy with the most recent type of apparatus was tried, but the speaker was unable to see the tumor because of its situation and the thick, short neck of the patient. As much force as he regarded as justified, both internally and externally, was used to bring the tumor into sight, but without avail. The man bore all this as well as could be expected, although complaining very bitterly about the pain and discomfort, especially as he had pyorrhea of all the teeth. In contradistinction to this maneuver, it was very brilliantly demonstrated that indirect laryngoscopy, in his particular case, at least, was very much to be preferred. The tumor was very easily removed on the first serious attempt, and there has been no recurrence. Histologically, it proved to be a fibrolipoma. Those who had examined the man could see that the place of attachment of the tumor is hardly to be discerned under the laryngoscope. He brought up the point regarding direct and indirect laryngoscopy to accentuate the fact that each method has its own particular field of application.

Suppurative Meningitis, Probably of Syphilitic Origin.

DR. NORVAL H. PIERCE: The patient was a male, laborer, aged fifty years. No tuberculosis or cancer in family history. Chancre fifteen years ago, but no secondary signs of syphilis developed. Past history showed measles at seven, followed

by corneal ulcer; also corneal ulcer (right) four years ago. No other illnesses. Present complaint began last April with pain behind right ear, accompanied by discharge. In August the ear became painful. The tympanic membrane apparently ruptured, and discharged pus for several days. Last September he began to have headaches, and in November he quit work. He had attacks of dizziness at short intervals. On December 5th he fell to the floor three times in the morning. From December 15th to 21st complained of frequent attacks of dizziness and headache. This continued, accompanied by loss of appetite and also loss of weight. As to his condition when he entered the clinic, he complained of slight headache and pain in the right ear. Paracentesis was performed by Dr. Lewy. He was admitted to the hospital on the 13th of December. Another paracentesis was performed by Dr. Smith, and followed by a slight purulent discharge, which stopped by February 4th. Was sent home, but returned to the hospital complaining of headache and dizziness. At this time examination of the right ear showed profuse purulent discharge; left, chronic catarrhal otitis media. On January 29th, white blood count, 12,300; hemoglobin, eighty per cent; albumin in urine; no sugar or casts. Hearing tests: Right ear, voice not heard; osseous and aerial conduction not heard. Left ear, whisper, fourteen feet; osseous conduction of an a^1 of five seconds; aerial conduction, fifteen seconds. Low limit for left ear, $G._2$ Weber test, to left, fifteen seconds. Nystagmus marked to left. Rotation: Eight seconds' nystagmus from diseased ear, and thirty seconds from left side. Cold irrigation of left, nystagmus to right on looking ahead. Pointing test showed deviation to left. No response to cold or heat in right ear. February 2, 1916, complained of severe pain in occiput and neck. Very dizzy. Cerebration low. White blood corpuscles, 6,400. February 3, 1916, Wassermann negative. Urine showed albumin, two per cent sugar; casts absent. He was given iodid of potassium at this time, which was continued practically up to the time of death. Chest and abdomen negative. Blood pressure, systolic, 140; diastolic, 70. Temperature, 100°; in morning, 99°. At no time was the temperature above 100°. Cerebration still low.

Examination of the reflexes showed pupils reacted to light

and accommodation slowly. Knee jerks weak. Babinski negative. Muscular strength slightly reduced on left side. Fundus examination unsatisfactory because of corneal opacity.

The relatives of the patient refused all operative procedures, but lumbar puncture was made on February 5th. Fluid under pressure. The opalescent spinal fluid showed albumin, 2.2 per cent. White blood cells, 787 per cubic millimeter. Bacteriologic content not reported.

February 6, 1916, slight stiffness of the neck. Slight Kernig on both sides. Marked spontaneous nystagmus to both sides—horizontal and slightly rotatory.

On February 8th the condition was unchanged. The patient went into deep coma and died on the 9th, at 4 a. m. On autopsy a very interesting temporal bone was found. A large mass of tissue, having the consistency of rubber, entirely occupied the jugular bulb. Its character could not be determined without microscopic examination. It might be an old organized clot. Its appearance would suggest that as the most probable thing. But there were no symptoms of sepsis at any time while he was under the speaker's observation. It might possibly be an organized gumma, but there were no symptoms of interference with cerebral circulation, when he was first seen at least, and we would expect that to be present if it were a gumma. However, that could only be ascertained by microscopic examination. Unfortunately, the cochlea was sawed through at autopsy, but it showed nothing macroscopically except that it was pretty well filled up with rather a homogeneous material, which probably resulted from the suppurative labyrinthitis. The temporal bone was perforated on its roof, and this condition again suggested syphilis, as the perforation went right straight through the upper portion of the bone into the sigmoid groove, whereas the perforation of the mastoid was posterior. This peculiar destruction of the bone would suggest a syphilitic process. It was hardly a well defined osteomyelitis. The lateral sinus contained no clot. There was fluid blood in it, which again interfered with almost any theory one might have regarding the growth in the jugular bulb.

The next point of interest was that in a case of suppurative meningitis the blood count might be as low as 6,200, with rather a lower fever—subfebrile—and absence of stiff neck. He com-

plained of headache toward the last, but it was not marked. He had none of the classical symptoms of a meningitis, and, in fact, the diagnosis made at the time before death was that there was some cerebral complication. The brain was preserved, cut, and shows very beautifully the manner in which the pus traveled along the arachnoid spaces. It shows how impossible it is to drain a suppurative meningitis. (Dr. Pierce showed the specimen.)

DISCUSSION.

DR. GEORGE E. SHAMBAUGH thought that the second case reported by Dr. Pierce permitted of some speculation as to the diagnosis. It seemed to him quite unlikely that a patient could be examined, even in a superficial way, and a suppuration of the labyrinth overlooked; in fact, not even the suppurative otitis media recognized. As a matter of fact, it may be very easy to overlook a chronic suppurative otitis media with a fistula in the region of Shrapnell's membrane. Such cases often give no history of discharge, and all that one detects is a dry scab, which is not easily removed and might very readily be mistaken for a little flake of cerumen. It is only after dislodging this scab that the fistula can be discovered.

It seemed to the speaker that it was extremely unlikely that a patient would develop the complication of suppurative labyrinthitis secondary to an otitis media, and not give very accurate symptoms pointing toward this lesion. The onset of a suppurative labyrinthitis is a tremendous shock to an individual, and is associated with very marked symptoms. In view of this fact, the speaker wondered whether the intracranial trouble in this case did not have its origin in syphilitic infection, as suggested by Dr. Pierce, and whether the otitis media, which was recognized the second time the patient was seen, was not an independent affair quite distinct from the fatal complication. The dead labyrinth has to be accounted for, but, as we all know, this is not such an uncommon occurrence, independent of any suppurative otitis media.

DR. J. HOLINGER thought the tumor seen in the bone had the typical consistency of a gumma.

DR. PIERCE said that the specimen had been in formalin for some time; it was not so gummy when first removed.

DR. HOLINGER said that the inner plate of the bone was rough and uneven—another peculiarity found in syphilis. The whole course of the labyrinth affection would also suggest this etiology. He thought the perforations from the sinuses, in different directions, gave a clear explanation that on account of the gumma in the bulb of the jugular vein the flow of blood was interfered with, and so was the collateral circulation.

DR. GEORGE W. BOOT did not agree with Dr. Holinger. It looked to him very much like an old organized thrombus in the jugular vein just below the bulb, because the process extended below the jugular bulb. The opening in the bone, aside from the perforation, caused by the disease, was an anomaly. Syphilitic meningitis is not apt to be suppurative.

DR. JOSEPH C. BECK was inclined to agree with Dr. Boot's diagnosis. He thought it was an old chronic suppuration. The patient being so long ill, his vitality would have been reduced to some extent, also his resistance, and there would be no possibility of a high blood count. He would like to know what the differential count showed.

By making a Wassermann test of the spinal fluid we have a most definite diagnosis of malignant syphilis. That should be done for differential diagnosis.

He saw the perforation in the specimen of the temporal bone, and the tumor, but did not examine it; however, he thought it was probably due to an old organized thrombus in the jugular bulb.

DR. J. R. FLETCHER wanted to call to Dr. Pierce's attention the fact that there was a break through the jugular bulb to the posterior fossa. He did not know whether Dr. Pierce had noticed this or not. That would make the condition somewhat of a problem. It seemed to him that it was an old thrombus, which caused this bone destruction during its active period. Meningitis or cerebellar abscess should, however, have developed at this time.

DR. PIERCE, in closing the discussion, said that the condition other than the meningitis did not look to him like a simple inflammatory one.

The orders were left to make a Wassermann on the spinal fluid, but through some mistake it was omitted.

There was no tenderness or swelling over the mastoid to indicate active inflammation within the mastoid.

He presented the specimen this evening simply as it came from the dead body. His next step will be to investigate the interior of the mastoid, and find whether there was a suppurative process within the mastoid.

Personally, his diagnosis as to the ear disease was syphilis, but, of course, this would not explain the whole picture. He died clearly of a frank, well defined, suppurative meningitis. But back of that there probably was a syphilitic process. There might be further data acquired when the labyrinth was sectioned.

Dr. Shambaugh thought the diagnosis might have been made when the patient first came to the hospital. If he had had any labyrinthitis before, or had it at that time, the speaker was not prepared to say, but at least, when he came to the hospital the first time, it was in a quiescent state, so that there were no symptoms of it. He gave no history of previous spells of dizziness, so that in the actual working of the clinic he was passed through with the diagnosis of an external otitis. The external auditory canal was very much swollen, and it was very difficult to get a view of the tympanic membrane. The whole thing will be largely cleared up by further investigation.

Giant Formation in Larynx Accompanying Hypophyseal Trouble.

Dr. Joseph C. Beck presented a man, sixty-five years of age, who came to him only a few days before, with laryngeal difficulty, which affected both speech and breathing. On examination, a large mass, about the size of the speaker's thumb, was found, which was soft, and fell into the larynx. The man's physiognomy has changed in the last year. The lips became very large; the tongue also became very large, and his head and features have enlarged. X-ray examination showed definite destruction of the sella turcica. The man had hemianopsia in one eye. The other eye was blind, but this was due to smallpox scars on the cornea.

The interesting point in this case was the occurrence of a laryngeal trouble in connection with hypophyseal disturbance. Such cases of giant formation in the larynx have not been described in the literature, so far as Dr. Beck knew. The patient was suspended that day, and the mass referred to could be moved. It looked to Dr. Beck like a hypertrophy of the mucous membrane, with relaxation.

Complete Destruction of Nose.

DR. GEORGE W. BOOT showed a case, which the members had seen before, of destruction of the nose, the septum and all the turbinates, in a man who had received an injury several years ago, and had been treated for syphilis. The man attributed the loss of the nose to the treatment, but it was probably due to syphilis. A year ago he had a strongly positive Wassermann reaction. He refused to have a new nose made. Vision was all he wanted improved, but that was impossible.

The interesting part of the case, aside from the destruction of the nose, was the manner in which the action of the soft palate could be seen when the patient spoke.

Lupus of the Nose.

DR. GEORGE W. BOOT: The second case came to the clinic at Rush the afternoon of the meeting. He was only sixteen years old; of Italian parentage, but born in England. The trouble had lasted for three days. He was treated in a London hospital, as he says, by the Finsen light, and under this treatment for nine months the condition of the nose cleared up entirely. Then he came to this country a year and a half ago, and the nasal trouble has recurred. The diagnosis made by one of the men in the skin clinic was tertiary lues; the speaker made one of lupus. The boy had a positive Wassermann reaction, two plus, but denied any knowledge of any venereal infection.

Probable Gumma of Frontal Sinuses.

DR. ROBERT SONNENSCHEIN reported the case of a man, aged twenty-nine years, who had a chancre in May, 1915, with general adenopathy. Shortly thereafter he had a very profuse, large maculopapular eruption. He developed severe pain and headaches. He was immediately put on inunctions of mercury and large doses of potassium iodid. He was also given nine intravenous injections of neosalvarsan. He had ninety mercurial injections altogether. The patient developed very severe pain in the region of the left frontal sinus; later, also in the right, and at present there is still pain in the right sinus, which is especially severe at night. Morphin has very little effect on the pain. At no time was any pus found in the nose,

although the middle meatus was opened and suction applied. Externally, nothing definite felt or seen, but tenderness over the frontal sinus, especially at the external angle. X-ray showed a peculiar circumscribed area in the extreme external end of the right frontal sinus. Just what that shadow means the roentgenologist could not say. Gumma had been suggested. Dr. Pierce suggested a possibility of a neurologic condition, and advised that the temperature be taken and a leucocyte count made. Any suggestions as to diagnosis and treatment would be gratefully received.

Paper: Surgical Anatomy of Frontal Sinus, With Demonstration of Anatomic Specimens.*

By George E. Shambaugh, M. D.

DISCUSSION.

Dr. Charles M. Robertson said that operation on the frontal sinus, by either the external or internal method, should be determined by skiagraph, and he agreed thoroughly with Dr. Shambaugh regarding the external operation. He has seen cases in which the ostium opened on the other side. For instance, the right frontal sinus had no opening on the right side of the nose, but on the left side, separately from the left frontal sinus. In nearly all of these cases a very small amount of drainage should be sufficient to cure the case, because if we have a small amount of cavity we have a small amount of pus, and if we have a small cavity, the cavity is filled very easily and the pus forced down into the nasofrontal duct. Those are the cases that get well of themselves. The cases that do not resolve are those in which the frontal sinus is extensive, as Dr. Shambaugh said, extending back over the orbit or out to the zygoma. He saw one cadaver in which the frontal sinus extended clear out into the zygomatic process and communicated with the cells of the mastoid through the zygoma. This was a pneumatic skull absolutely; the two mastoid processes communicated through the occipital bone, and this, too, was the only case that he had ever seen in which the frontal sinuses were above a very large and numerous set of eth-

*See page 707.

moidal cells that extended clear around the orbit, so that there was a partition between the ethmoidal cells and the antrum of Highmore. If anyone had attempted to cure an empyema in that case by either external or internal operation, he certainly would not have succeeded. It would have been absolutely impossible to have gotten drainage from all of those cells.

At present, the internal operation is the one of selection. The ethmoidal cells that push up usually open into the nose by separate openings, although, as Dr. Shambaugh said, some of these ethmoidal cells have one frontal sinus opening into them. The whole business is an ethmoidal cell pushed up into the frontal sinus; the antrum of Highmore is an ethmoidal cell pushed down into the superior maxillary bone. They are a combination of cells that are thrown in to give the skull less density, less weight, and also for a resonating cavity, and in each case the cells differ. Dr. J. C. Beck several years ago said that these skulls were usually bilateral in their configuration. We find that is not so in the frontal sinus, and very often not so in the ethmoidal cells, one sinus being very large, extending out to the absolute edge of the zygoma, and the other one very small, or no cell at all.

The matter of external operation with deformity has never frightened him as much as some others, because in doing it he has always left a broad bridge, in which the deformity is absolutely at a minimum. Experience has taught him that very many of these ostia rather hug the cribriform plate, more than was indicated by Dr. Shambaugh. In fact, in nearly all the cases that he probes, the direction is more upward, forward and inward than outward.

Dr. Otto T. Freer thinks it helps the anatomic conception of the frontal sinus and of its relation to the ethmoid cells to appreciate that the sinus itself is merely an ethmoid cell that has invaded the substance of the frontal bone, as the other ethmoid cells are inclined to do, in the manner, as Vacher says, of a bubble blown into glass. The penetration of other ethmoid cells into the frontal bone is made evident by looking at the border of the ethmoid notch of the frontal bone where are seen the depressions or foveolæ which complete the uppermost cells of the lateral masses of the ethmoid bone. In many specimens it may be seen that the foremost of these ethmoidal

foveolæ has entered into the frontal bone to form a large cell lying just posterior to the frontal sinus, having the same general form as the sinus, and often extending laterally beyond it, such cell being called a "bulla frontalis." These anatomic facts are clearly shown in Figures 1 and 2 in Dr. Freer's recent article, "The Frontal Sinus: Opening It Through the Nose."

The structural features mentioned by Dr. Shambaugh have definite anatomic designations which Dr. Freer wished to see in general use, in order to produce a clearer mutual understanding in discussing the intricate anatomy of the frontal sinus region.

Dr. Shambaugh spoke of the lower outlet of the nasofrontal duct as being found either in the infundibulum or in front of it, under the anterior end of the middle turbinate. In the first case the outlet is called an "ostium infundibulare," in the second an "ostium recessuale," as it opens directly into the hollow extending upward towards the frontal sinus under the middle turbinate, a space called the "recessus frontalis" by Killian.

The prolongation of the frontal sinus backward alongside the cribriform plate, mentioned by Dr. Shambaugh, is called a "recessus paracribrosus" by L. Onodi. Where the cribriform plate and the olfactory fossa, of which this plate forms the floor, project forward enough to create a bulge in the posterior wall of the frontal sinus, the projection into the sinus so formed is called a "torus olfactorius" by L. Onodi. The wall of such a torus not only projects strongly into the sinus on the inner posterior part of the circumference of the sinus outlet (ostium frontale), but this wall is often very thin, so that Herbert Tilley, in describing the intranasal opening of the frontal sinus, warns against enlargement of the ostium inwardly, because the cranial cavity might be opened here.

Very small frontal sinuses, mentioned by Dr. Shambaugh, are common in women with smooth foreheads lacking frontal eminences. Such sinuses may be mere slit-like cavities lying just above the brow. To such faces, as a group, Dr. John Gordon Wilson has given the name of "monkey-face type." In spite of their small size, little sinuses, if chronically suppurating, may cause anterior orbital abscesses by creating caries of the floor of the sinus.

Hajek calls attention in his work on the anatomy of the sinuses to the difficulties met with in probing the frontal sinus, and shows that the false direction apt to be given the probe is mainly due to the lower border of the middle turbinate, which directs the probe too far outward, involving danger of pushing a stiff probe into the orbit. Where the probe, therefore, will not readily slip up into the sinus, the middle turbinate should be resected anteriorly before further attempts at probing are made, care being taken in all probing never to use force.

The prevalent idea that chronic frontal sinus suppuration cannot get well unless the mucous membrane lining of the sinus be curetted away is as mistaken in regard to the frontal sinus as it is in regard to the maxillary antrum. Only rarely, where caries exists, is the mucosa so degenerated that it cannot recover its normal state, if good intranasal ventilation and drainage be secured for the sinus. This is the view of Max Halle, the most experienced and radical of the intranasal frontal sinus operators. Curettage of a noncollapsible cavity with rigid bony walls cannot have the beneficial results offered by curettage of a cavity whose walls are composed of soft parts. In the latter case, curettage establishes healthy granulations which enable the walls of the collapsing cavity to grow together, obliterating it. In a bony cavity no such collapse can take place, and curettage replaces epithelium-lined mucous membrane, capable of recovery, with a granulating surface, which merely sprouts exuberantly and continues to suppurate.

Dr. George W. Boot showed a specimen with a very large frontal sinus divided by numerous septa that were cut off in making the preparation. The one peculiarity of this sinus was that while most of the septa ran in an anteroposterior direction, there was still another running in a transverse direction, and behind it a large cell, in which he could place a hook.

Dr. Norval H. Pierce reported a case, of which he was reminded by one of the specimens. A young woman had an intranasal operation. The case was characterized by great pain for some length of time. The intranasal operation gave little or no relief, and then she was operated externally. The pain continued, but abated for some time, when she came to

the speaker's notice with a swelling over the left lateral orbital region, fluctuating temperature, and he advised external operation. The wound, which had been very small, was enlarged, and he opened into an abscess cavity at the extreme outer portion of a frontal cavity that resembled the one he referred to, shown by Dr. Shambaugh, to a nicety. The septum had not been removed in operating. The mesial portion of the cavity was quite normal, except that the internal wall was missing, leaving the dura exposed. This lateral portion (indicating) was filled with granulations, and when this septum was taken away, as the speaker did in his operation, and the granulation tissue curetted out, she made a favorable recovery.

Paper: The Oral Cavity in Relation to Ear, Nose and Throat.*

By T. E. Carmody, M. D.

DISCUSSION.

Dr. J. R. Fletcher thought the paper illustrated very forcibly how much we fail to see. We are very liable to neglect the mouth. He recalled a case seen some time ago in his clinic. The patient complained of severe pain in the throat. Examination by several clinicians had revealed nothing. The throat was perfectly normal, but a large abscess had formed around the roots of the posterior superior molar on the right side. Opening that abscess relieved the throat pain. Frequently, in Dr. Fletcher's experience, when the cause of complaint cannot be found elsewhere, he finds it in the mouth. Another case, referred by a dentist, came to his mind. All of the teeth had been extracted years ago. A fistula developed at the side of the upper left middle incisor, which seemed by probing to come from the antrum. At least the last of four dentists to whom the lady had been thought so. Upon examination there seemed to be a large exostosis protruding into the nose from the floor. He thought this might be an unerupted tooth which, upon operation, proved to be the fact. The fistula was a communication between the nose and mouth, around the tooth root.

*See page 682.

Dr. Fletcher thought the subject timely and exceedingly interesting.

Dr. P. J. H. Farrell said he had heard one of our noted neurologists read a paper upon neuralgia, and the speaker took exception to the statement that in his very wide experience he had never seen a case that could be diagnosed as neuralgia resulting from any condition of the teeth. Dr. Farrell instanced a case where there was an impacted third molar that apparently was the cause of the neuralgia. The patient had this unerupted third molar removed, and was entirely relieved of a very serious neuralgia for a period of three or four months. The neurologist said he did not care—that he had seen some thousands of cases—and to Dr. Farrell's disappointment he saw this case in a few months with a return of his neuralgia as severe as ever.

The speaker had had a number of cases since that time which were similar, and he has come to believe that if the diagnosis is made of neuralgia—a typical tic douloureux—the teeth may be eliminated as causative factors. There is a difference between neuralgia and the pain from an abscess. The dental profession is in the habit of calling any pain in the gums, jaws or teeth "neuralgia," nowadays. Neurologists all agree, the speaker believes, in eliminating the teeth as a cause of neuralgia.

Dr. Joseph C. Beck said that probably Dr. Carmody was able to speak so authoritatively on the subject because he had practiced dentistry before he became an otolaryngologist, and consequently had perhaps seen some of his own mistakes, or those of others, which had been shown so beautifully in the skiagrams. It has been the speaker's misfortune not to get the cooperation of dentists in doing this work. He has had considerable difficulty in trying to exclude a nasal or sinus condition, believing it was caused by the teeth, and asking the dentist to make an examination. The dentist would say that he had been taking care of the teeth, and that there was nothing wrong, sending the patient back to Dr. Beck. Finally, after the patient had put up with this for some time, he would consult another dentist, perhaps at Dr. Beck's instigation, and find just such conditions as those brought out by the essayist.

The speaker said something about every hospital, or perhaps every number of men practicing together having a dentist for their own cooperation. That is Dr. Beck's practice, which he adopted on account of the difficulty experienced.

Dr. Beck cited a case of a young woman who had a nosebleed that he could not stop. He suspected the teeth, sent her to a dentist, who said the teeth were all right. A skiagram showed definite disease. The patient was sent back to the dentist, who told her that what she needed was a good nose doctor. However, there was a dead root held by a small bridge, and not only that, but a sequestrum above that; the superior maxilla had a fistula leading to the middle of the nose, near the sinus, which was the cause of the nosebleed from suppuration.

Another point he wished to make was with regard to the X-rays. The films that are taken by dentists in order to show a clear picture of the absorption of bone in suppuration about apical infections do not assist him as much as does a dental picture that has been recommended by Eisen—who was killed in Milwaukee recently—who made X-ray pictures of half the side of the face to show the antrum and both jaws. Pictures taken in this way give a very good illustration of these diseases in connection with antrum trouble, and the speaker believes that that is the way these skiagrams help in diagnosis.

Dr. Beck was exceedingly glad to have heard Dr. Carmody's paper.

DR. S. A. FRIEDBERG asked Dr. Carmody whether any other treatment than extraction was of benefit in cases of small abscesses.

DR. N. SCHOOLMAN asked Dr. Carmody whether the finding of teeth in the antrum of Highmore, especially in the case reported as subsequently being found to be a sarcoma, would be conceded as a dermoid cyst, or whether these teeth are found in any other way.

DR. CHARLES M. ROBERTSON mentioned a condition of the mouth that is of interest to nose and throat men, and has considerable connection with the teeth, namely, the subject of leukoplakia. He wished Dr. Carmody would say something

about the occurrence of leukoplakia, as regards teeth, in which carious or roughened teeth are the primal cause of the leukoplakia. We are interested in leukoplakia for the reason that it breaks down into epithelioma, and from epithelioma into carcinoma. Dr. Robertson has seen numerous cases of cancer of the tongue, of the cheek and of the back part of the throat that were directly caused by rough or carious teeth, either from a bad plate that is anchored on floating teeth, or from pyorrhea teeth that are ragged-edged and irritate the mouth. Leukoplakia used to be considered as a disease accompanying irritation in the shape of smoking and chewing of tobacco. That, in Dr. Robertson's opinion, to a certain extent, is a mistaken idea. He believes that the teeth were the real cause of the leukoplakia, and not the irritation caused by the fumes of tobacco or the chewing of tobacco, as in nearly all these cases the patients were excessive chewers or smokers and also had ragged, rough teeth, with pyorrhea pockets.

The essayist did not bring out very strongly the idea of stretching the palate for the straightening of the septum. In nasal surgery, as it is practiced today, submucous operations are produced on children of tender years, which septums could probably be straightened by the broadening of the palate, as that brings the septum down, whereas it was a V-shape before, and this elongates the vertical dimensions of the septum, pulling it into a straight plane.

DR. H. KAHN asked Dr. Carmody at how late an age one may begin to broaden the jaw in septal cases.

DR. CARMODY, in closing the discussion, said that Dr. Fletcher spoke of extracting all the teeth, and Dr. Farrell also spoke of the extraction of teeth for neuralgia. The cases of real tic douloureux that we see very frequently have had all of the teeth extracted, and it has been found that in those cases the pain is usually located in one tooth. The patient will point out which tooth it is in. As soon as the tooth is extracted, the patient will locate the pain in the next tooth, and keep on until they are all extracted. What Dr. Farrell said is true—those cases do not clear up, if they are true neuralgias, by extraction of the teeth alone. If they are treated generally and brought to a good physical condition, the neuralgic condition will prob-

ably disappear, but not because of the extraction of the teeth.

Dr. Beck spoke of his experience, and it has been the speaker's also, only probably not to quite so great an extent, because of the dentists with whom he is associated all know that he was a dentist, and when he tells them what to do for a case, they generally do it. However, he has had the very experience Dr. Beck related. Dr. Beck referred to the taking of pictures, but Dr. Carmody did not know exactly what plates he referred to. However, he has found that the plates, so far as the jaws and teeth are concerned, are unsatisfactory. They do not give the idea of a root very well. They, however, give a very general idea of the sinuses and jaws.

Dr. Friedberg asked whether extraction alone is necessary. In many of the cases, that is all that is necessary, because cavities will drain very well. In others, however, it is necessary to curette the bone, because while we have not a true necrosis there, as we understand it—death of the bone en masse—we have a caries of the bone, and the little fine particles have to be taken out.

Dr. Friedberg asked if every tooth in which there was an abscess should be extracted. Dr. Carmody said that in many cases there is quite an abscess cavity in the tooth. If the dentist can open through the root canal well enough and treat it, thus getting rid of the infection, and then fill it properly, the tooth would not have to be extracted, but if there is any amount of caries which cannot be gotten rid of, extraction is necessary.

Dr. Fletcher asked if the very modern dentists do not actually go through the soft parts into the bone. Dr. Carmody replied that dentists do this very frequently, and cut off the end of the root. Very frequently he makes a fistulous opening. In that way he simply drains the cavity from both ways, and in the case of a lower tooth it is very convenient to do this, because you can get better drainage then through the tooth.

As regards dermoid cysts, the probabilities are that a dentigerous cyst is very similar to a dermoid cyst; however, it is not a change like we have in dermoids. In the dermoid, of course, we have the development of hair and teeth, while in

the case of dentigerous cyst we simply have, as a rule, a part from one of the other teeth, or a normal tooth which has gotten into the maxillary sinus, or into a region where it cannot erupt itself, and is forced in there. The irritation probably has something to do with the production of the cystic fluid.

Dr. Robertson spoke about leukoplakia, and Dr. Carmody believed that if he would look up all the literature on the subject, he would find that smoking has something to do with it, nevertheless. There is no question but what teeth have a great deal to do with epithelioma in the mouth. Rough teeth very frequently produce epithelioma without leukoplakia at all. He has seen one case of socalled leukoplakia of the lips where there were no teeth present, and had not been for a number of years. The leukoplakia occurred after the removal of the teeth. That was not said in denial of Dr. Robertson's statement, but that is not the opinion of most people. Dr. Carmody believes, however, that most of the cases of epithelioma of the mouth are due to either bad plates, bad bridges, broken-down teeth, or something of that kind, and the smoking is just another irritant. Frequently epithelioma of the lip is not due only to smoking, but to a displacement of some cells in shaving at times, because of cutting of the lips, etc., that probably forces in some epithelium into the lower stratum of the lip. That, however, was only his own idea, and he did not know whether it was really so or not.

Dr. Kahn spoke of broadening of the arches. That was simply mentioned by him in the paper, because it had been covered in so many cases. Nevertheless, he believes that a great many septa can be straightened in this way, and the little patients saved a great deal of trouble.

Another thing to be remembered is that the lymphatics in younger patients are open more than in older patients. The speaker had the misfortune to have a patient die of meningitis following a submucous resection, and since that time he has hesitated to operate on young children. This patient was only eleven years of age. Someone asked how late these operations could be performed. The oldest was twenty-eight years. The probabilities are that spreading the arch and helping to straighten the septum, whether by drawing it down forcibly

or simple stimulation of the growth of the bone of the palate, meets with better success if done earlier. Orthodontists believe now in beginning with the temporary set of teeth, if there seems to be trouble, and especially after the eruption of the six-year molars. Those are erupted very often before the fifth year, and regulation of the teeth can be begun at that time, so as to straighten the teeth, and, of course, it will help the nose a great deal more than if done later.